Exhibit 108

UNITED STATES DISTRICT COURT
DISTRICT OF NEW JERSEY

IN RE JOHNSON & JOHNSON TALCUM POWDER PRODUCTS MARKETING, SALES PRACTICES, AND PRODUCTS LIABILITY LITIGATION ***THIS DOCUMENT RELATES TO ALL CASES***	**MDL NO. 16-2738 (FLW) (LHG)**

RULE 26 EXPERT REPORT OF
JUDITH ZELIKOFF, PHD

Date: November 16, 2018

Judith Zelikoff, PhD

I. BACKGROUND AND QUALIFICATIONS

I received my Ph.D in Experimental Pathology and Immunology at Rutgers: NJ Medical School (formerly known as University of Medicine and Dentistry of NJ) in 1982, after receiving a Master's degree from Fairleigh Dickinson University in Microbiology. My post-doctoral training was in toxicology at the NYU School of Medicine, Department of Environmental Medicine as a National Heart Lung Blood Institute (NHLBI) fellow.

I am currently a tenured-professor in Toxicology at NYU. As part of the NYU NIEHS (National Institute of Environmental Health Science) Center of Excellence, I serve as Director of the Community Engagement Core. In this capacity, I engage with environmentally-impacted underserved communities throughout New Jersey and New York to better engage the community to achieve long-term and sustainable outcomes, processes, relationships, discourse, decision-making, and implementation regarding environmental health. These goals are carried out through town hall meetings, focus groups, listening sessions, forums on relevant environmental concerns, surveys, as well as outdoor and indoor measurements of toxic metals such as lead, cadmium, mercury, and arsenic in water, air, and soil. I also provide service to the NYU School of Medicine as a member of the Grievance Committee, Institutional Animal and Use Committee (IACUC) and as an NYU Senator representing the School of Medcine.

I have served in numerous leadership positions in the field of toxicology, including NIH Study Sections, United Nations Environmental Programme, NASA boards, and National Academy of Science Panels (i.e., Institute of Medicine, National Research Council and Engineering, and Medicine's Board on Earth Sciences and Resources), as well as Environmental Protection Agency study sections and advisory boards concerning the toxic effects of air pollution, metals, and alternative tobacco products. Furthermore, I served for two years (2010-2012) as a member of the National Toxicology Program (NTP) Board of Scientific Advisors. In this capacity, I reviewed documents and provided input and guidance on the toxicity of various chemicals that were nominated for review and sent to the NTP for study and/or discussion. In some cases, we also decided on the carcinogenicity of specific compounds. I was not part of the NTP 10 ROC or 12 ROC, both of which deferred the decision on talc.

In addition, I presented about 150 international/national papers in the areas of toxicology and environmental and public health. I have organized several international toxicology meetings, served as editor for several toxicology/environmental public health books and authored numerous book chapters in the same areas. I have over 125 publications and book chapters in the area of immunotoxicology (for which I received a Lifetime Achievement Award from the Society of Toxicology), air pollution toxicology, metal toxicology, immunotoxicology, and developmental and reproductive toxicology associated with inhaled metals, mixtures, nanomaterials, dusts (i.e., World Trade Center Dust), and tobacco/nicotine toxicology.

I have held numerous executive positions in the Society of Toxicology (SOT) which includes three years as Secretary on the SOT Executive Council and one year as Chair of the Education Committee and Committee for Diversity Initiatives Committees. I have also provided leadership for four individual SOT Specialty Sections (SS). I have served as President of the Immunotoxicology, Metals and Ethical, Legal, Forensic and Societal Issues Specialty Section and currently serve as Senior Councilor of the Inhalation and Respiratory Specialty Section. I have received three major SOT awards including the Mentorship Award from "Women in Toxicology", Global Host award and in 2018, Education award for meritorious teaching skills in toxicology. As a teaching scholar, I have taught and continue to teach toxicology on a global level in such countries as Thailand, Nigeria, South Africa, Tasmania and New Zealand.

My education, training and publications are further set out in my Curriculum Vitae, which is attached to this report as an **Exhibit A**.

II. MANDATE AND METHODOLOGY

Mandate: I was asked to review the scientific literature and assess whether there is a biologically plausible explanation for the increased risk of ovarian cancer with the perineal use of talcum powder products.

The notion of biological plausibility is multi-factoral. As a part of my analysis, while considering the totality of the evidence, I evaluated the genital use of talcum powder products, the routes of exposure by which talcum powder could reach the ovaries, the composition of the talcum powder products, the biological and toxicological effects of talcum powder, and the potential mechanisms of carciogenisis. Biological plausibility does not mean proof of mechanism, but rather whether what is known about the products is consistent with a cause and effect relationship.

I performed an independent, comprehensive literature review using research databases and search enginges including PubMed, ToxLit and Google to identify relevant literature. The keywords/phrases used initially for searching, included: talc, talcum powder, talc and cancer, talc and toxicity, talc and toxicology, ovarian cancer, oxidative stress, talc and ovarian cancer, animal models and talc, talc powder and the immune response and talc chemical structure. Keywords and phrases expanded upon those terms in later searches.

More than 300 publications (research papers, reviews, abstracts, reports, documents) and book chapters from the 1960s to the present were identified as having some relevancy for the talc-ovarian cancer topic. Following closer scrutiny of these publications, between 200-250 research papers, scholarly reviews, abstracts, documents, reports were found critical for informing my opinion. Toxicological studies, including *in vivo, in vitro* and *ex vivo* investigations, were the topics most appropriate for my area of expertise. In addition, I have reviewed depositions and numerous documents, internal memorandum

and published and unpublished studies and testing results that I have found in my own searches, documents provided by attorneys, and documents that I requested. A list of materials and data considered for this report are attached as **Exhitit B**.

My opinions below are based upon my experience as a toxicologist and research scientist and have been reached through employing the same scientific methodology and rigor that I employ in my academic research and professional duties. To my knowledge, I considered and evaluated the majority of all available relevant studies in the process of evaluating the literature, including those that reported an elevated risk of ovarian cancer with exposure to talc and those where other chemicals were reported within talc-based body powders, including those that did not find an increased risk. The same approach was used in evaluating the animal data and the mechanistic data.

III. TALC

Primary talc deposits are found on almost every continent around the world[1]. Talc is commonly formed by the hydrothermal alteration of magnesium- and iron-rich rocks (ultramafic rocks) and by low-grade thermal metamorphism of siliceous dolomites. Talc is the softest mineral on earth, mined around the world for use in a wide variety of products personal, cosmetic or industrial in nature. The word "talc" can refer to two things. The first is a mineral and the second is a commercially available product that can be used both industrially and in pharmaceuticals and cosmetics. For this report, when talking about the former, I use the term "mineral talc," and when talking about the latter, I use the term "talcum powder products." Johnson & Johnson talcum powder products are classified as cosmetic talc. Dermal contact (including perineal application of talcum powder products) is a primary route of human exposure, while inhalation also represents a route of exposure for talc/talcum powder products.

As a mineral, talc corresponds to the chemical structure of hydrous magnesium silicate with a formula of $Mg_3Si_4O_{10}(OH)_2$ and a theoretical chemical composition, expressed as oxides, of 31.7% by weight magnesium oxide (MgO), 63.5% silicon dioxide (SiO_2) and 4.8% water (H_2O). Talc belongs to the silicate subclass phyllosilicates and is known as a sheet silicate. It is the softest mineral on Mohs' hardness scale, and it's structure and chemical bond arrangement is such that it is easily broken into thin sheets. The structure consists of three sheets that are octahedrally coordinated magnesium hydroxide groups (brucite layer) layered between 2 layers of tetrahedrally linked silica layers. The apical oxygen atom positions of the tetrahedral layers are shared with one of the oxygen atom positions of the octahedral layer. The composite sheets repeat every 9.4 angstroms and the triple-sheet crystalline units are held together by van der Waals forces. Talc particles are normally plate-like in shape, but may form mineral fibers, as discussed below.

[1] https://minerals.usgs.gov/minerals/pubs/commodity/talc/mcs-2017-talc.pdf

Small amounts of aluminum and ferric (III) iron can substitute for silicon in talc tetrahedral sites. Trace amounts of nickel and small to moderate amounts of ferrous (II) and ferric (III) iron, aluminum and/or manganese can substitute for magnesium in talc octahedral sites. Additionally, talc deposits may contain varying amounts of quartz, nickel, chromium and cobalt, as well as asbestos or asbestos-forming minerals including amphibole (tremolite, actinolite, antigorite and anthophyllite) and serpentine (chrysotile) (Cralley, 1968; Lockey, 1981; McCarthy 2006; Rohl, 1976). The pH of cosmetic talcs are usually alkaline (8.0-9.5) and are insoluble in water, cold acids or in alkalis.

Talc powder particle size depends on the process used to make the powder. Johnson and Johnson's analysis of particle size in talcum powder shows particles range on average from 0.8 µm to over 50 µm, with a median particle size of 11.39 µm, where approximately 43.9% of particles are less than 10 µm (JNJTALC00878141).

A. Fibrous Talc

As a mineral, talc is most commonly found in plate-like form, but may also form as true mineral fibers that are asbestiform (IARC 2010, IARC 2012). Asbestiform talc (also known as fibrous talc) is different from talc containing asbestos. Fibrous talc fibers are very long and thin and occur in parallel bundles that are easily separated from each other by hand pressure (IARC Monographs, 2010). The 2010 IARC clearly states that the term 'asbestiform fiber' means any mineral, including talc, when it grows into an asbestiform habit. In its fibrous form, talc has been classified as a Group I, known carcinogen (IARC 1987 Supp 7; IARC 2010; IARC 2012). OSHA considered fibrous talc exposure limits to be equivalent to those of asbestos (OSHA, 1972). In 2010, IARC expanded the Group 1 designation ("known carcinogen") from "talc containing asbestiform fibers" to "talc containing asbestos or other asbestiform fibres." (IARC, 2010). Additionally, the American Conference of Governmental Industrial Hygienists (ACGIH) clarifies that "talc may also take the form of long thin fibers (fibrous talc) and can occur in bundles that are easily separated (asbestiform talc). Asbestiform talc should not be confused with talc containing asbestos…" (ACGIH, 2010).

Asbestiform talc fibers have been reported by Johnson & Johnson and Imerys to be found in: mines from which ore for Johnson & Johnson talcum powder products were sourced; in talcum powder used in Johnson & Johnson talcum powder products; and in the Johnson & Johnson talcum powder final product.[2]

Recent TEM testing on historic samples of Johnson's Baby Powder from 1978 showed the presence of fibrous talc in the product (Longo & Rigler, Feb 2018 MAS Report). Additional TEM testing of 30 samples of J & J baby powder and Shower to Shower dating from a span of many years resulted in a finding of fibrous talc in 15 samples (Longo & Rigler, Aug 2017 Expert Report).

[2] See also: IMERYS477879 (fibrous talc in Grade 66 Q1 composite); JNJ 000269848 (talc needles found in medicated powder 1971, see with TEM results in JNJ 000281921); JNJ 000245002 (Fibrous talc in Hammondsville mine 1970)) .

IV. ASBESTOS

Asbestos, like talc, is a naturally occurring silicate mineral, but with a different crystal structure (Mossman & Churg, 1998). Asbestos is a generic name referring to a group of naturally occurring mineral silicate fibers. It is recognized as a known human carcinogen by the U.S. Occupational Safety and Health Administration (OSHA), the U.S. Environmental Protection Agency (USEPA) and the National Toxicology Program (NTP)(OSHA, 2014; USEPA, 1995; NTP, 2016). The National Institute for Occupational Health (NIOSH) has stated there is no safe level of asbestos and the American Conference of Governmental Industrial Hygienists (ACGIH) characterizes it as a "confirmed human carcinogen" (NIOSH, 1980; ACGIH, 2017). All forms of asbestos are Group 1 carcinogens (carcinogenic to humans)(IARC, 2012).

The U.S. EPA defines asbestos by limiting the term to 6 specific fibrous minerals from two distinct groups: chrysotile (from the Serpentine group); and amosite, crocidolite, tremolite, actinolite and anthophyllite (from the Amphibole group). "Asbestiform" describes the pattern of growth of a mineral that is referred to as a "habit" (IARC, 2010). Minerals with a "non-asbestiform" habit have crystals that grow in two or three dimensions, and "cleave into fragments, rather than breaking into fibrils" (*Id.*). Chrysotile occurs in the asbestiform habit, whereas, of the amphiboles, amosite and crocidolite occur only in the asbestiform habit, and tremolite, anthophyllite and actinolyte can occur in asbestiform or non-asbestiform habits. OSHA defines an asbestos fiber as having a length > 5mm and a length:width aspect ratio of 3:1, whereas the USEPA definitition incorporates the aspect ratio of > 5:1 (OSHA, 1992; USEPA, 1987).

While amphibole and serpentine asbestos may have fibrous habits, they have very different forms. The amphiboles are double-chain silicates also called inosilicates. The basic structural unit is $(Si_4O_{11})^{-6}$ with side groups that are responsible for the overall amphibole structure. Amphiboles are distinguished from one another by the amount and positioning of metal atoms including: sodium, calcium, manganese, magnesium, iron(II), iron(III) and aluminum. Traces of these types of asbestos are extracted when other minerals are being mined and, due to inefficient or non-existant separation techniques, are ultimately incorporated into the final product. Even incidental contamination by amphibole forms of asbestos is hazardous enough to cause asbestos-related illnesses (Rohl & Langer, 1976).

The serpentine group of minerals has the formula $Mg_3Si_2O_5(OH)_4$ and the structure resembles a bending sheet. Chrysotile is the only one in which the sheets are bent to form continuous tubes, which gives the mineral the fibrous habit related to asbestos. Chrysotile is very flexible and less likely to be "friable" than the amphiboles. Friability of asbestos is generally defined as the ability to easily be turned into a dust with finger pressure. It is this friability that can release asbestos fibers and potentially result in health problems.

A. Asbestos in Talc

Associated minerals found in commercial talc products vary from deposit to deposit depending on the formation conditions. The most common minerals associated with talc include chlorite, magnetite, dolomite, calcite, mica, quartz and fluoapatite (Fiume et al., 2015). In its natural form, some talc also contains asbestos, classified as a Group I, "known carcinogen" by IARC (IARC Monographs, 1973, 1977, 1987, 2012). Amphiboles and serpentine fibers have been associated with many talc deposits (Van Gosen, 2004; Marconi and Verdel, 1990; Lockey, 1981; Rohl and Langer, 1974; Gamble et al., 1979; Kleinfeld et al., 1973, 1974; Pooley, 1972 (JNJ000319762); Chidester, 1968). The close proximity of asbestos and talc in mineral deposits makes extraction of either material alone difficult, if not impossible. (Rohl and Langer, 1974; IARC, 2010; Dion et al. 2010[3]).

Cralley (1968) analyzed twenty-two commercially available cosmetic talcum products (manufacturers not reported). Authors reported the fiber content ranged from 8% - 30% (by count) with an average of 19% and that the fibrous material was predominantly fibrous talc. Pooley and Rowlands (1975) analyzed twenty-seven talc powders (cosmetic and industrial) and detected tremolite fibers in three samples.

Because asbestos is a known carcinogen, its presence in cosmetic talc is unacceptable (FDA, 2012; FDA 2015). The former Director of National Institute for Occupational Safety and Health (NIOSH) and former President of Industrial Minerals Association – North America (IMA-NA) stated in a recent deposition that if there were a fiber of asbestos in talcum-based products it would "certainly" provide a biologically plausible mechanism for increased lung disease, and that he suspected it would also have a "similar mechanism of disease in other tissues and organs" (Deposition of Robert Glenn, October 18, 2018, 341:15-342:3).

In 1976, specifications were developed for cosmetic talc requiring that no detectable fibrous, asbestos mineral be present (CTFA, 1990; Fiume, 2015). The talc industry, and specifically Defendants, developed a "zero tolerance" standard for asbestos in talc (IMERYS 170006; JNJ 000383662; JNJ 000001918). Despite this standard, the presence of asbestos in cosmetic talc has been reported in the literature, and Johnsom and Johnson indicated in a letter in 1973 that "asbestos-form particles cannot be removed from talc" and that the "Johnson & Johnson process for beneficiating Vermont talc…will not guarantee a zero tolerance for elongated particles" (JNJ 000233691). In 1976, Rohl et al. tested 20 different talcs and powders including 20 body powders, baby powders, facial talcums, and also one pharmaceutical talc to determine their mineralogical and chemical composition. Where known, all were formulated prior to 1973. Of the 20 products, 9 contained detectable amounts of tremolite and anthophyllite, principally asbestiform, while some also contained fragmented forms of these minerals. The amounts ranged from tenths of a percent to over 14% by weight; two contained detectable amounts of chrysotile asbestos fiber. Eight samples contained quartz, seven ranging from 2 to 5%, with one as high as 35%. Analyses showed that the consumer products examined were rarely the pure mineral talc, but rather were mixtures of various minerals.

[3] Available online at: http://www.irsst.qc.ca/media/documents/PubIRSST/R-755.pdf

In 1984, Paoletti et al. performed studies by electron microscopy to assess asbestos contamination in industrial and cosmetic talcs from the Italian market and the European Pharmacopoeia (Paoletti, 1984). Nine of the 25 pharmaceutical and cosmetic grade talcs contained tremolite fibers, with total percent asbestos concentrations ranging from 0.4% - 22%. About half of the talc powders revealed the presence of asbestos: in five samples chrysotile (a serpentine asbestos) was present, the others contained tremolite and anthophyllite (an amphibole asbestos).

Cosmetic and pharmaceutical talc products from deposits in Vermont, Montana, North Carolina and Alabama were examined and tested positive for asbestos (Blount, 1991). The investigator of that study recently affirmed the samples included Johnson & Johnson baby powder, purchased off the shelf (Deposition of Alice Blount, PhD, April 13, 2018). The early analytical methods used to measure asbestos fibers before 1990 were not very sensitive and thus it appears that extrapolation of the levels of asbestos from counts measured before this date could have been conservative (Blount, 1991).

In a study that examined the amphibole asbestos content of commercial talc deposits in the USA, Van Gosen et al. (2004) found that the talc-forming environment directly influenced the amphibole and amphibole-asbestos content of the talc deposit. Specifically, the study found that contact metamorphic talcs showed a strong tendency to contain amphiboles, and regional metamorphic talc bodies consistently contained amphiboles, which display a variety of compositions and habits (including asbestiform). In a German study (Mattenklott, 2007), the author examined the presence of asbestos in talc powder and found that in one-quarter of the 57 talc powder samples tested, asbestos could be detected. Two samples contained quantities exceeding 0.1 weight percent which could reach a value of 10,000 fibers/m^3. This weight percent is, in some cases, half that reported by Johnson & Johnson in their internal documents, as seen in the corporate depositions reported below.

Defendants have claimed that asbestos has been "eliminated" from cosmetic talc products.[4] However, there is substantial evidence that talcum powder products still contain asbestos, recognized as a Group 1 carcinogen. During the recent deposition of John Hopkins (Johnson and Johnson corporate representative), Mr. Hopkins affirmed testing results showing the presence of asbestos in mines from which talc ore was taken for use in Johnson & Johnson baby powder products, processed talc used in Johnson & Johnson baby powder products, and in complete Johnson & Johnson baby powder products. Those results may be found at Exhibit 28[5] of Dr. Hopkins' deposition. Additional examples of testing performed by and commissioned by Johnson and Johnson and Imerys may be found at Exhibit 47 to the deposition of Julie Pier, corporate representative of Imerys.[6]

In 1975, McCrone Associates also confirmed the presence of amphibole particles, alone and in bundles as seen in Defendants' internal documents (JNJMX68_000012745). In 2004, a television station reported that Johnson's Baby Powder had been analyzed and found anthophyllite asbestos at 0.2% (JNJ 000089413). A 1972 Johnson & Johnson document demonstrates the presence of up to 5% chrysotile in

[4] PCPC Submission to FDA, July 2009 – "Since the early 1970's, the relevant industries voluntarily eliminated asbestos contamination from talc products."
[5] Ex. 28, John Hopkins Dep. (Aug. 16 & 17, 2018; Oct. 17, 2018; and Nov. 5, 2018).
[6] Ex. 47, Julie Pier Dep. (Sept. 12 & 13, 2018).

Johnson's Baby Powder and Shower to Shower samples (JNJ 000232996). These data clearly demonstrate the possibility for women who used talcum powder during these dates to have had exposure to this ovarian carcinogen.

Recent TEM testing on historic samples of Johnson & Johnson baby powder from 1978 showed the presence of fibrous anthophyllite in the product. (Longo and Rigler, 2018; Ex. 47, Pier Dep.). Additional TEM testing of 30 samples of Johnson & Johnson baby powder and Shower to Shower ranging in production date over a span of many years resulted in a finding of amphibole asbestos (tremolite, anthophyllite, richterite and actinolite) in 17 samples. (Longo and Rigler, 2017). Additionally, I have reviewed a recent report prepared by Dr. William Longo and Dr. Mark Rigler that reports that talcum powder products manufactured by Johnson & Johnson's Baby Powder and Shower to Shower have contained and continue to contain asbestos and talc containing asbestiform fibers (e.g. talc occurring in a fibrous habit).[7] These results were obtained from testing talcum powder product samples manufactured during the period of the 1960s through the 1990s. Results showed 37 of 56 samples tested contained tremolite and/or anthophyllite asbestos, and 41 of 42 samples tested contained fibrous talc.

The substantial evidence of the presence of asbestos and fibrous talc in talcum powder products provides a biologically plausible explanation for the increased risk of ovarian cancer associated with the perineal use of talcum powder products.

V. HEAVY METALS

A. Properties of Heavy Metals

Nickel is classified by IARC as a human carcinogen (Group 1) (IARC, 1973, 1976, 1979, 1982, 1987, 1990). The exact mechanisms of nickel-induced carcinogenesis are not known, but likely involve genetic and epigenetic routes. Nickel (II)-induced genotoxicity may be aggravated through the generation of DNA-damaging reactive oxygen species (ROS) and the inhibition of DNA repair by this metal. Nickel exposure also causes a broad spectrum of epigenetic effects. Contact with nickel compounds can cause a variety of adverse effects on human health (Zambelli and Ciurli, 2013).

Nickel ions have been shown to cause single-strand DNA breaks and DNA-protein crosslinks (Patierno, 1985). In a study by Patierno (1985), Chinese hamster ovary cells were exposed to $NiCl_2$, and nickel-induced DA-protein crosslinking appeared in late S phase of the cell cycle (*Id.*). Authors associate these alterations as an early event in the process of nickel transformation (*Id.*).

Contact with nickel compounds can cause a variety of adverse effects on human health, such as nickel allergy in the form of contact dermatitis, lung fibrosis, cardiovascular and kidney diseases and

[7] Expert Report of William E. Longo, PhD and Mark W. Rigler, PhD (Nov. 14, 2018).

cancer of the respiratory tract. Chronic non-cancer health effects may result from long-term exposure to relatively low concentrations of pollutants (Duda-Chodak and Blaszcyk, 2008). Although the accumulation of nickel in the body through chronic exposure can lead to a number of diseases, the most serious concerns relate to nickel's carcinogenic activity. Increased risks of malignant tumors, such as nasal and sinusoidal cancers, and cancers of the lung and larynx have been noted (IARC, 1987). The marked differences in the carcinogenic activities of various nickel compounds most likely reflect the differences in their uptake, transport, distribution and retention, and ultimately—the capacity to deliver nickel (II) ions to specific cells and target molecules.

In experimental animals, nickel compounds induce tumors at virtually all sites of application (Denkhaus, 2002; IARC, 1987; Zabmelli, 2013). The routes of administration that were shown to produce tumors include inhalation, intramuscular, intrarenal, intraperitoneal, intraocular, subcutaneous and the intra-articular space (*Id.*).

Chromium is a naturally occurring element found in rocks, animals, plants, soil, and volcanic dust and gases. It comes in several different forms, including trivalent chromium (chromium (III)) and hexavalent chromium (chromium (VI)). In contrast, chromium (VI) compounds cause cancer in humans and in experimental animals and exert genetic toxicity in bacteria and in mammalian cells *in vitro* (Fang, 2014; IARC, 2009). Adverse health effects, other than cancer, associated with chromium (VI) exposure include occupational asthma, eye irritation and damage, perforated eardrums, respiratory irritation, kidney damage, liver damage, pulmonary congestion and edema, upper abdominal pain, nose irritation and damage, respiratory cancer, skin irritation, and erosion and discoloration of the teeth. Some people with extensive dermal exposure can also develop an allergic skin reaction, called allergic contact dermatitis (Bruynzeel et al., 1988). Primary irritant dermatitis is related to the direct cytotoxic properties of chromium, while allergic contact dermatitis is an inflammatory response mediated by the immune system. During reduction to the trivalent form, chromium may interact with cellular macromolecules, including DNA (Wiegand et al., 1985), or may be slowly released from the cell. Complexes of chromium (III) that are bound to lower molecular weight ligands are most likely to be able to traverse cell membranes.

Chromium (III) has weak cell membrane permeability, allowing it to cross the cell membrane, where it can bind to DNA and cause lesions, resulting in genetic damage such as strand breaks and DNA-protein crosslinks (Nickens, 2010). This damage leads to genomic instability. Another study has shown that chromium (III) causes DNA damage in cells by interfering with base pair stacking in the cell's replication cycle, and chromium (VI) intercalates DNA – both directly cause genotoxicity in vivo (Fang, 2014).

Hexavalent chromium compounds are classified by IARC as carcinogenic to humans (Group 1)(IARC, 2009). Mechanistically, they have been shown to cause direct DNA damage after intracellular reduction to Cr(III), mutation, genomic inistability, aneuploidy, and cell transformation (*Id.*). Chromium (VI) can cause damage leading to dysfunctional DNA replication, aberrant cell cycle, DNA strand breaks, dysfunctional DNA repair and DNA-protein crosslinks and directly causing genotoxicity (Nickens, 2010).

Besides direct genotoxic effects of chromium (VI), chromium compounds such as chromate can activate transcription factors involved in inflammation and tumor growth (IARC, 1990). Major factors

governing the toxicity of chromium compounds are oxidation state and solubility. These compounds, which are powerful oxidizing agents and thus tend to be irritating and corrosive, appear to be much more toxic systemically than chromium (III) compounds, given similar amounts and solubilities. Chromium (VI) enters many types of cells and, under physiological conditions, can be reduced by hydrogen peroxide (H_2O_2), glutathione (GSH) reductase and ascorbic acid to produce reactive intermediates, including chromium (V), chromium (IV), thiyl radicals, hydroxyl radicals, and ultimately, chromium (III). Any of these species could attack DNA, proteins and membrane lipids, thereby disrupting cellular integrity and functions (De Mattia, Bravi *et al.* 2004). Besides cancer, chromium is one of the most common skin sensitizers. It also causes toxicity of the kidney, liver, gastrointestinal tract, and cardiovascular, hematological and reproductive systems along with causing developmental effects.[8] High doses of chromium (VI) compounds have been reported to cause developmental toxicity in mice and shown to potentiate the effects of other toxicants, including the nephrotoxins, mercuric chloride, citrinin, hexachlorobutadiene, and maleic acid.

Cobalt IARC declared that cobalt metal with tungsten carbide is *probably carcinogenic to humans (Group 2A)*, while cobalt metal without tungsten carbide is *possibly carcinogenic to humans (Group 2B)*. Two different mechanisms of genotoxicity, (1) DNA breakage induced by cobalt metal and especially hard metal particles, and (2) inhibition of DNA repair by cobalt (II) ions contribute to the carcinogenic potential of cobalt compounds (Lison et al., 2001; IARC, 2006). Cobalt can also contribute to allergic reactions. In humans, gastrointestinal absorption of cobalt has been reported to vary between 5 and 45% and it has been suggested that absorption is higher in women than in men. Cobalt can be absorbed through intact human skin (IARC, 2006). Soluble cobalt salts interfere adversely with cell division, bind irreversibly to nucleic acids in the cell nucleus, induce chromosome aberrations in plants, and are weakly mutagenic in some *in vitro* tests. Injections or implantation of cobalt metal, alloys and compounds induced local and sometimes metastasizing sarcomas in rats, rabbits, and mice (*Id.*). Data indicating possible carcinogenic effects of cobalt alloys or compounds in human populations has arisen from medical use, use in hard-metal industries, and from cobalt production sites.

B. Metals in Talcum Powder Products

In an early paper by Cralley et al., (1968), 22 cosmetic talcum products purchased off the shelf were analyzed for fibrous content, selected metals and quartz. In these studies, 19 samples contained cobalt under 25 parts per million (ppm) by weight, chromium under 22 ppm, nickel below 29 ppm and manganese under 78 ppm. Certain samples had a nickel content of 1270 ppm, chromium 340 ppm and 1210 ppm nickel; qualitative tests demonstrated that some of the chromium was hexavalent (carcinogenic form). All of these talcs had a considerable fiber content (suggesting the presence of asbestos) (*Id.*). Studies here suggest that women who used talcum powder in the 1960s could have been exposed to considerable amounts of toxic heavy metals depending on the type of talc used and frequency of use (*Id.*).

[8] Accessible online at: https://www.atsdr.cdc.gov/csem/csem.asp?csem=10&po=10

In a 2013 study by Rehman, toxic and carcinogenic heavy metals were found to be present in small amounts in all 30 brands of cosmetic talcum powder tested; the concentrations of heavy metals differed dramatically depending upon the brand of talcum powder (Rehman, 2013). Heavy metals measured (and found in samples) included cadmium, chromium, copper, cobalt and lead. Authors found all levels to be within safe limits. However, authors caution that excess use of talcum powder affects the health of the consumer (*Id.*).

In a paper by Gondal et al. (2012), published in Applied Optics, lead and chromium were measured in talcum powder using laser-breakdown spectroscopy. Using this system, the authors were able to detect 15-20 parts per million (ppm) of lead and 20-30 ppm of total chromium in the talcum powder sample. This study, like that by Rehman, demonstrates the presence of toxic heavy metals associated with talcum powder. However, the levels of heavy metals in this study were significantly higher. The method used for measuring metals in this study was far more precise than that used by Rehman et al. (2013). This study supports the presence of toxic and potentially carcinogenic metals in some talcum powders.

According to Johnson & Johnson's corporate representative, the maximum amount of allowable nickel in the company's talcum powder products was 5 ppm (Deposition of John Hopkins, August 16, 2018, Ex. 3). Written specifications state that the maximum allowable nickel content is 10 ppm (JNJ 000629320; JNJ000488188; JNJMX68_000022920). Despite these limits, nickel in concentrations exceeding 2000 ppm were reported in Vermont talc used in talcum powder products for decades, greatly in excess of the product specification limit of 10 ppm (JNJ 000629320; JNJ 000488188; JNJMX68_000022920). Examples of testing results for heavy metals in Defendants' talcum powder products can be found in **Exhibit C**, attached to this report.

Over the years from 1972 to 2004, talc mined in Vermont had consistent, excessive levels of nickel, routinely exceeding 94 to 250 times the upper limit provided in J&J's specifications (Exhibit C). This is troubling considering nickel is a known carcinogen (IARC 2012).

Cobalt was found in Vermont talc ores in amounts ranging from 8 – 89 ppm from 1972 through 2004. Like nickel it, too, appears to occur routinely in talc products in amounts exceeding the 10 ppm upper limit for heavy metals in the talc product specifications (Exhibit C).

Internal documents outline Johnson & Johnson's concern regarding the potential carcinogenic nature of chromium (VI), a Group I carcinogen (JNJ 000131758; JNJ 000131754; JNJ 000378044; JNJ 000378046). A 2010 J&J memo written discusses raising the upper limit acceptable for total Cr to 7 ppm (JNJ 000131758). An accompanying memo also discusses the relationship between chromium (III) and chromium (VI) (JNJ 000131754), and a discussion of the inhalation of hexavalent chromium is contained in this document. Regardless of valence, Grade 66 analyses consistently show total chromium contents far in excess of 5-, 7-, or 10 ppm. During the period from 1972 thru 2004, the chromium content varied from 25 ppm to 569 ppm (Ex. 47, Pier Dep.), with typical levels around 200 ppm.

Interestingly, there is a significant difference between the reported chromium content of Grade 66 talc when the sample has been prepared by Johnson & Johnson (internal) method BPT 148 versus the

United States Pharmacopeia (USP) method which uses a total digestion technique (IMERYS-A_0015621). The levels reported using the USP method were much higher than the Johnson & Johnson method (*Id.*).

C. Fragrances

There are more than 150 different chemicals added to Johnson's Baby Powder and Shower to Shower products. I reviewed the expert report from Dr. Michael Crowley that concludes that some of these chemicals may contribute to the inflammatory response, toxicity, and potential carcinogenicity of Johnson & Johnson's talcum powder products.[9] I concur with his opinion.

There is substantial evidence that talcum powder products contain excess levels of nickel, chromium, and cobalt, all known carcinogens and/or inflammatory agents. Moreover, a significant number of the fragrance chemicals added to talc elicit an inflammatory response. Each of these elements individually and together can contribute to an inflammatory response caused by the product. As will be explained in more detail below, inflammation is a known mediator of ovarian cancer. The presence of these inflammatory agents provides additional biologic evidence explaining the causal relationship between genital use of talc and ovarian cancer.

VI. EXPOSURE – TALC PARTICLE ACCESS TO THE BODY

A. Exposure Routes

Based on the tenets of toxicology, there are four basic routes of human exposure including: inhalation, ingestion, dermal and injection.

A common exposure route for cosmetic talc is via the dermal route, including vaginally after perineal application. Talc body powders are often applied to the perineum for hygienic purposes. It has been shown that glove powder and other materials can migrate upwards through the female reproductive tract (Venter & Iturralde, 1979; Iturre and Venter, 1981; Sjosten et al., 2004; Heller et al., 1995) and the data are supported by animal investigations (Wright et al., 1996; Edelstam et al., 1997; De Boer, 1972; Henderson et al., 1986), also reflective of a dermal exposure route.

Inhalation is the route of exposure that has been most commonly studied to assess talc toxicity. In one inhalation study, after talc exposure of hamsters, there was a consistent elevation in cytotoxic enzyme levels, and macrophage phagocytosis was persistently depressed (Beck et al., 1987). These results also indicated that, when a similar mass of talc and granite dust (12% quartz) was deposited in the lungs,

[9] Expert Report of Michael Crowley, PhD (Nov. 12, 2018).

talc caused more lung injury than did granite (*Id.*). Based on its physical properties talc, in a powder form, can be inhaled while being applied (EPA, 1992; IARC, 2010). Additional evidence that application of talc body powder products results in inhalation exposure of talcum powder is provided in a 2017 study by Longo, et. al., and other studies (Longo, September 2017, "*Below the Waist Application of Johnson & Johnson Baby Powder*"; Wells, 1979; van Huisstede, 2010; Frank and Jorge, 2011; Jasuja, 2017).

1. Dermal - Migration Through the Upper Genital Tract

Animal models: Though animal studies have limitations due to the differences in anatomy, they provide evidence that talc can migrate through the reproductive system. Rats were exposed vaginally or via the perineum to either talc or no treatment for 3-mo on a daily basis (Keskin et al., 2009). In this study, there was evidence of foreign body reaction and genital infection, along with an increase in inflammatory cells in all the genital tissues. While no neoplastic changes were observed, the number of ovarian follicles in the talc groups were increased. No peritoneal changes were observed. The investigators concluded that talc by perineum exposure has adverse effects on the genital system in the form of foreign body reactions and infection (*Id.*).

In a series of two experiments, Henderson et al. (1986) demonstrated the presence of talc in the ovaries of two groups of animals following vaginal and intrauterine talc applications, whereas none was present in the ovaries of control animals. Particles were also seen in animals that had received intravaginal talc that were sacrificed after 4 days. (*Id.*)

Studies by Wright et al. (1995) also demonstrated the potential toxicity of retrograde uterine passage of particulate matter. Despite the aforementioned studies which demonstrate the plausibility of talc translocation, a study by Wehner et al. (1996) failed to demonstrate the same outcomes in a small sample of monkeys, which may have been due to the small sample size.

Human studies: A number of human studies over many years have observed migration of particles following vaginal administration: these studies began as early as 1961 when Egli and Newton studied the translocation of carbon particles following vagina application. In 1972, De Boer deposited colloidal carbon black (CB) suspension in the uterus, cervical canal or vagina in over 100 patients prior to surgery (De Boer, 1972). Subsequent observation revealed rapid translocation of CB to the oviducts and beyond. Some CB deposited in the cervical canal also translocated to the uterine passage, albeit in a lower percentage of patients (*Id.*). An early study by the National Institute of Occupational Safety and Health (NIOSH) in 1972 showed commercially available talc body powder samples contained fibers, and that exposure to fibers occurred during diapering (JNJ 000231304).

A study by Venter and Itteralde (1979) administered radiolabeled human albumin microspheres (no size provided) in the vagina of patients, followed by surgical removal of uterus, oviducts and ovaries. Results demonstrated that 9 out of 14 patients had radioactivity in their oviducts and ovaries. Recent studies have demonstrated the presence of talc particles in ovarian tumors (to be discussed in a later section). Another clinical study examined a total of 24 women undergoing oopherectormy (Heller et al.,

1995). In this case, women were questioned as to their use of perineal talc applications. Ovarian tissue was removed from each group and analyzed and quantitated for talc by polarized light and electron microscopy. These data support the ability of talc to migrate from the perineal region upward and reach the upper genital tract (*Id.*).

Further evidence for migration of particles to the upper genital areas comes from a document from the FDA to Dr. Epstein (Cancer Prevention Coalition, University of Illinois, Chicago) concerning Citizen Petitions dated 1994 and 2008 and requesting a cancer warning on cosmetic talc products. In this document, the FDA stated that “the potential for particulates to migrate from the perineum and vagina to the peritoneal cavity is indisputable” (JNJ 000488318).

In addition, a 2004 document from Luzenac America to Dr. Al Wehner (IMERYS 137677) recalls a 2004 published paper by Sjosten et al. (2004). Luzenac states that the paper “offers some compelling evidence **in support** of the ‘migration’ hypothesis.” The paper concluded that starch particles migrate from the vagina through the Fallopian tubes up to four days after examination with powdered gloves (*Id.*). The author of the Luzenac document goes on to state that combining this evidence with the theory that talc inititates epithelial inflammation and you have a “potential formula” for the NTP classification of talc as a carcinogen.

The most recent systematic review of the association between genital use of talcum powder products and ovarian cancer (Penninkilampi, 2018) reported an increased risk of ovarian cancer with increased perineal talcum powder use, with a slightly higher risk in women who report greater usage. Data was collected as “lifetime” usage – frequency of use over time. Any use was associated with increased risk of ovarian cancer as compared to no use, and women with long-term (> 10 years) talcum powder use had an increased risk. The authors concluded perineal talcum powder use and ovarian cancer were consistently associated, with a slightly higher risk in women who report greater usage.

Pathways that allow for the migration of particles to the lymph nodes are also available for that complex portion of the lymphatic system surrounding the ovaries. Importantly, studies by Chan et al. (2007) have demonstrated a positive association between lymphadenectomy and survival in stage 1 ovarian cancer patients. In support of this finding, Cramer et al. (2007) described the presence of talc particles in pelvic lymph nodes of a woman with ovarian cancer and long-term genital exposure to cosmetic talc.

Animal and human studies demonstrate that talcum powder products can migrate from the perineal region to the ovaries.

2. Inhalation

Effects of size on particle translocation and toxicity have been studied most extensively with inhaled particulate air pollutants and nanomaterials. These studies will be discussed to provide a scientific

premise for movement of particles of a certain size throughout the body. Small-sized particles can enter the bloodstream – translocation of particles and often toxicity are related to their size; perhaps because of the larger mass concentration of smaller vs. larger particles (Driscoll et al., 1997).
J&J's analysis of particle size in talcum powder products shows particles range on average from 0.8 µm to over 50 µm, with a median particle size of 11.39 µm, where approximately 43.9% of particles are less than 10 µm (JNJTALC00878141).

Ultrafine particles (UFPs; < 0.1 µm) can directly affect the cardiovascular system by migration from the respiratory system to the systemic circulation (Nakane, 2012; Elder et al., 2006; Kreyling et al., 2006). Inhaled UFPs deposited in the lung can pass through the epithelial barrier because of their very small size; some particles may move into lung capillaries and then into the systemic circulation. Numerous studies and reviews have been written concerning the migration of these particles. In a systematic literature review (Nakane, 2012), particle size was shown to be a strong factor for migration. Particles that were translocated to various sites were observed to have the following sizes: ≤0.05 µm for remote organs, ≤1 µm for blood, and ≤10 µm for lung tissues. In order to be detected in the blood, particles that have passed through the epithelial barrier of the lungs must migrate into the capillaries. The largest chance for migration to the brain was observed at a 0.05-µm cutoff size. However, MnO_2 particles as large as 1.3 µm have also been detected in the cerebral cortex (Nakane, 2012). A categorical regression analysis based on currently available inhalation data showed that all of the effects of particle size, particle material, animal species, and exposure route were statistically significant (*Id.*). The effects were large for particle size and particle material, and small for exposure route and animal species. These results suggest that, in an experiment to evaluate the migration of solid particles, the characteristics of the particles (i.e., size and material) should be considered carefully.

Evidence from an internal document (1971) demonstrates rolled talc fibers between 0.1 - 3µm in a Johnson and Johnson's commercial product (JNJAZ55_000005957). Other documents from Defendants have demonstrated that while median particle size is ~10.5 µm, sizes can be as small as 0.3 µm (IMERYS030347; IMERYS031791). V66 non-shear talc was approved for use in JNJ Shower to Showder products and the size of some of the particles had a diameter as small as 0.1 µm (JNJTALC000878141). While the median particle size was ~12 µm, the standard deviation was very high (~9µm) demonstrating a large range of particle sizes. Fine-size particles such as those found in talc, can also translocate readily throughout the body (Peters et al., 2006), providing a strong basis for the ability of fine-size talc particles (<2.5 µm to migrate throughout the body).

Ultrafine and fine particles can penetrate through the different tissue compartments of the lungs and eventually reach the capillaries and circulating cells. These particles are then translocated by the circulation to other organs including the liver, the spleen, the kidneys, the heart and the brain, and the ovaries where they may be deposited. It remains to be shown by which mechanism(s) ultrafine particles penetrate through tissue and enter capillaries. Lymph capillaries remove the large protein molecules and other particulate matter from the tissue spaces of the lung. Thus, cellular debris and foreign particles inhaled into the lungs can be conveyed to the regional lymph nodes.

Talc particle size analyses for many inhalation studies demonstrated that most talc particles were between 1 and 8 µm; 1 µm is considered ultrafine in size and thus particles could easily migrate from the lungs and throughout the body. Genofre et al., (2009) examined the effect of talc particle size on induced pleurodesis following intrapleural injection of rabbits with two different sizes of talc. One group contained mixed sizes of talc (mean size = 25.4 µm) and the other group small size talc only (mean size = 4.2 µm with 50% <6.4 µm) (*Id.*). Particles of both sizes migrated to the spleen, liver and kidney; more small talc particles (compared to mixed talc) was seen in the liver and kidneys. Both size particles produced an acute systemic inflammatory response, with small particle talc producing a more pronounced pleural and systemic response and resulting in greater particle deposition in the organs than the mixed talc (*Id.*). In addition, serum levels of the pro-inflammatory cytokine, IL-8 and VEGF were more markedly increased in the small talc group (*Id.*). Particles found in all systemic organs were <5µm. A number of other studies have shown migration of talc particles from the pleural cavity to the systemic circulation (Ferrer, 2002; Rossi, 2010). It appears that small particles may be more easily taken up by the lymphatics than larger particles. The inflammatory effects observed showed a strong correlation with the small particle group. This study shows that size of talc particles matter and the smaller the size the greater the ability to translocate and increase the extent of the inflammatory response. As Defendants' internal documents demonstrate their talc particle size to cover a wide size range (100 µm to ~0.3 µm).[10], there is extensive evidence that particles can be inhaled and transported through the blood and lymph to the ovaries.

In 1993, the National Toxicology Program (NTP) issued a report from a study concluding that there was "some evidence of carcinogenic activity" in male rats, "clear evidence of carcinogenic activity" in female rats, and no evidence of carcinogenic activity in male or female mice exposed to aersols of talc reported as nonasbestiform cosmetic-grade (National Toxicology Program, 1993). Authors of that study speculated these effects could be due to cytokines released from macrophages or a nonspecific effect of the stress of inflammation (*Id.*).

In another study, rabbits were injected with normal size talc (Dmax = 8.36 µm) or larger particles talc (Dmax = 12 µm) (Ferrer et al., 2002). Pleural inflammation was greater with normal talc than large talc, and animals receiving normal talc had talc particles in the liver, supporting the premise that talc particles instilled into the pleural cavity can escape and migrate to extrapleural organs. Talc dissemination can be significant, and granulomas have been seen to develop in the interstitium after particles migrate from the lungs, with resultant pulmonary interstitial fibrosis (Hollinger, 1990). In another study illustrating talc dissemination (Werebe, 1999), talc was administered into the pleural space of rats. At both 24- and 48-hours, talc crystals were found in every organ of all animals, with the amount of talc being statistically different between the organs. Authors concluded there was a rapid absorption of talc through the pleural surface and a progressive systemic distribution of particles (*Id.*).

In addition to migration of ultrafine particles through tissue and movement to the lymhph nodes, fine and coarse particles may be phagocytized by macrophages and dendritic cells which may carry the particles to lymph nodes in the lung or to those closely associated with the lungs (IARC, 2010). The uptake of fine particles (0.1–2.5 µm in diameter) by macrophages is a specific ligand-receptor mediated

[10] IMERYS346016; IMERYS030347; IMERYS031791; JNJAZ55_000005957.

actin-based process (phagocytosis), whereas the uptake of ultrafine particles (<0.1 μm in diameter) apparently occurs by other, non-specific mechanisms (Peters, 2006). These mechanisms are termed "adhesive interactions," and include electrostatic, van der Waals and steric inter-actions (*Id.*). Particles with a diameter of 0.2 μm and smaller appear to enter cells passively, that is by a mechanism which is different from phagocytosis. Larger particles are much more avidly taken up by macrophages, but by the specific receptor mediated, actin-dependent mechanism. Below the particle size of 0.2 μm, particles increasingly enter the macrophages by the non-specific "adhesive interaction" mechanisms mentioned above (*Id.*).

There is substantial evidence in the scientific and medical literature that support a conclusion that talc powder particles can reach the ovaries through inhalation.

VII. MECHANISM OF CANCER

A. Cancer - General

Tumorigenesis, the formation and growth of tumors, is a complex and multifactorial progressive process of transformation of normal cells into malignant ones (Pogribny and Rusyn, 2014). It is characterized by the accumulation of multiple cancer-specific heritable phenotypes, including persistent proliferative signaling, resistance to cell death, evasion of growth suppression, replicative immortality, inflammatory response, deregulation of energy metabolism, genomic instability, induction of angiogenesis, and activation of invasion ultimately resulting in metastases. It encompasses genetic, behavioral, and environmental factors that can all contribute to its development.

Mutations can occur as a result of the processes inside the cell, or alternatively, can be caused by external factors, such as chemicals. In addition, some people can inherit faults in particular genes that make them more likely to develop cancer. While normal cells obey signals indicating they have reached their growth limit, in cancer cells, the normal signaling system is disrupted. Mutations in particular genes may result in over- or under- production of proteins, or the production of abnormally formed proteins, all of which can lead to a lack of cellular regulation.

In general, cancer is an uncontrolled growth of abnormal cells in the body, which occurs when the body's normal control mechanisms are disrupted. Excessive cellular division leads to a growth called a tumor. Mutations can happen by chance when a cell is dividing. Some mutations act by inhibiting normal controls over cell growth, leading to uncontrolled cell division. DNA may be damaged during routine cellular processes, and cells have mechanisms to repair that damage. However, over time, the damage may accumulate. Once cells exhibit increased cell growth, they are more likely to pick up additional mutations and are less likely to be able to repair the damaged genes.

If the DNA damage cannot be repaired, the cell can self-destruct, a process called apoptosis. In cancer cells, molecules in the repair pathway are faulty. For example, a protein called p53 normally determines whether genes can be repaired or if the cell should undergo apoptosis. Many cancers have a defective version of p53, and don't repair themselves properly. Thus, cancer cells can override self-destruct signals and don't undergo apoptosis when they should.

B. Genetic Mutations

Inherited mutations are passed down from parent to child and are present throughout a person's life in virtually every cell in the body. These mutations are also called germline mutations because they are present in the parent's egg or sperm (germ) cells. When an egg and a sperm cell unite, the resulting fertilized egg cell receives DNA from both parents. If this DNA has a mutation, the child that grows from the fertilized egg will have the mutation in each of his or her cells.

A genetic predisposition (sometimes also called genetic susceptibility) is an increased likelihood of developing a particular disease based on a person's genetic makeup. A genetic predisposition results from specific genetic variations that are often inherited from a parent. These genetic changes contribute to the development of a disease, but do not directly cause it. For example, mutations in the *BRCA* gene result in an increased risk for ovarian cancer. Some people with a predisposing genetic variation will never get the disease while others will, even within the same family. Genetic variations can have large or small effects on the likelihood of developing a particular disease. Although each of these variations only slightly increases a person's risk, having changes in several different genes may combine to increase disease risk significantly. Changes in many genes, each with a small effect, may underlie susceptibility to many common diseases, including cancer.

In people with a genetic predisposition, the risk of disease can depend on multiple factors in addition to an identified genetic change. These include other genetic factors (sometimes called modifiers) as well as lifestyle and environmental factors. Diseases that are caused by a combination of factors are described as multifactorial. Most disease-causing gene mutations are uncommon in the general population. However, other genetic changes occur more frequently. Genetic alterations that occur in more than 1 percent of the population are called polymorphisms.

Acquired (or somatic) mutations occur at some time during a person's life and are present only in certain cells, not in every cell in the body. These changes can be caused by environmental factors such as ultraviolet radiation from the sun, chemical exposure, or can occur if an error is made as DNA copies itself during cell division. Acquired mutations in somatic cells (other than sperm and egg cells) cannot be passed to the next generation.

Environmental and occupational exposures to natural substances, as well as man-made chemical and physical agents, play a causative role in human cancer. Acquisition of cancer-specific alterations may be triggered by the mutational and/or non-mutational (i.e., epigenetic) events in the genome which, in turn, affect gene expression and downstream phenotypes including persistent proliferative signaling, resistance to cell death, evasion of growth suppression, replicative immortality, inflammatory response,

deregulation of energy metabolism, genomic instability, induction of angiogenesis, and activation of invasion ultimately resulting in metastases.

Genotoxic carcinogens are agents that interact directly or after metabolic activation with DNA, causing mutations and leading to tumor formation. Non-genotoxic carcinogens are a diverse group of chemical compounds that are known to cause tumors by mechanisms other than direct damage to DNA. In a broad sense, carcinogenesis may be induced through either genotoxic or non-genotoxic mechanisms. However, both genotoxic and non-genotoxic carcinogens also cause prominent epigenetic changes (Pogribny and Rusyn, 2013). Disruption of epigenetic processes can lead to altered gene function and malignant cell transformation. Global changes in the epigenetic landscape are a hallmark of cancer.

The presence of talc particles in the ovaries (deep in the tumor) of some ovarian cancer patients and presence of talc in pelvic lymph nodes provides indirect evidence for talc carcinogenicity (Heller et al., 1996). Changes in signal transduction pathways that lead to increased and chronic inflammation are also associated with cancer, as are changes in cancer stem cells which have the ability to generate tumors through the processes of self-renewal and differentiation into multiple cell types. Cancer stem cells are thought to play a major role in tumor escape, chemoresistance/recurrence of ovarian cancer. Users of talcum powder have lower plasma levels of anti-MUC1 antibodies than non-users (Karageorgi et al., 2010). MUC1 is a protein highly expressed by ovarian, breast, and endometrial tumors, and low levels of anti-MUC1 antibodies are associated with poorer prognosis. Reducing immunity to MUC-1 could be one mechanism by which talc increases endometrial and/or ovarian cancer risk (Karageorgi et al. 2010).

C. Ovarian Cancer

There are two major categories of ovarian carcinogenesis based on the idea that tumors are heterogeneous: high-grade malignancies that tend to be fast growing and chemo-sensitive, and low-grade neoplasms which typically grow slowly, but are less sensitive to chemotherapy. The low-grade pathway is associated with a stepwise mutation process, whereas the high-grade develops through genetic instability (Lengyel, 2010). Ovarian cancer comprises at least five distinct histological subtypes, the most common and well-studied being high-grade serous ovarian cancer. The majority of these tumors arise from the distal end of the fallopian tube and evolve from premalignant lesions called tubal intraepithelial carcinoma (Saad, 2010). Several risk factors have been associated with increased risk of ovarian cancer and include: low parity, infertility, early age of menarche and late age of menopause.

Multiple mechanisms can explain the progression of ovarian cancer (Fleming et al., 2006; Fathalla, 2013; Saad, 2010; Smith and Xu, 2008). These mechanisms include: incessant ovulation- whereby repeated damage and trauma to the ovarian epithelium during ovulation increases the risk for genetic mutation and ovarian neoplasm during epithelium repair; pituitary gonadotropin changes- high levels of gonadotropins increase estrogen stimulation which can cause ovarian epithelial cells to become entrapped in inclusion cysts that undergo malignant changes; androgen/progesterone alterations- androgens stimulate ovarian cancer formation and progestins are protective; inflammation- factors that predispose to inflammation, such as endometriosis, PID, perineal talc use and hyperthyroidism could stimulate ovarian cancer. The molecular pathway in the inflammatory process involves intracellular

effectors implicated in malignant transformation such as VEGF, NF-κB, nitric oxide synthase, and cyclooxygenase (Williams et al., 1999).

Genetic mutations also play a role in the development of ovarian cancer. For example, certain mutations in the *BRCA1* or *BRCA2* genes increase a person's risk of developing ovarian cancer. Both inherited and acquired gene mutations work together to cause cancer. Even if one has inherited a genetic mutation that predisposes one to cancer, that doesn't mean he or she is certain to get cancer. Rather, one or more additional gene mutations may be needed to cause cancer. The inherited gene mutation could instead make one more likely to develop cancer when exposed to certain cancer-causing substances.

D. Roles of the Immune System

It is well established that inflammation has paradoxical roles during tumor development (Coussens and Werb, 2002). While acute inflammation can be protective against tumors, chronic inflammation provides an environment for the tumor to thrive. The net outcome of tumor-associated inflammation depends on the dominance of either tumor-promoting or tumor-suppressive actions. Inflammation normally functions to maintain tissue homeostasis in response to tissue stressors such as infection or tissue damage. However, studies also suggest a close association between inflammation and tumorigenesis (Rakoff-Nahoum, 2006).

Two stages of inflammation exist, acute and chronic inflammation (Ingersoll, 2011). Acute inflammation is an initial stage of inflammation (innate immunity), which is mediated through the activation of the immune system. This type of inflammation persists only for a short time and is usually beneficial for the host. Acute inflammation (e.g., involving innate immunity, macrophages, natural killer cells, neutrophils) frequently precedes the development of protective adaptive immune responses to pathogens and cancer.

Chronic inflammation, by contrast, has been shown to contribute to tumorigenesis at all stages (Crusz and Balkwill, 2015). It contributes to cancer promotion by inducing cellular proliferation; and to cancer progression by enhancing angiogenesis and tissue invasion. Over time, chronic inflammation can cause DNA damage and lead to cancer. Inflammation initiated by genital application of talc is likely to be sustained, since studies indicate that women start using talcum powder at an early age and continue using it for decades.

E. Ovarian Cancer and Inflammation

Inflammation plays an important role in the progression of ovarian cancer, and it is a biologically plausible mechanism that mediates ovarian cancer. Recent clinical and prospective data suggest that C-reactive protein (CRP), a marker of global inflammation, is associated with increased ovarian cancer risk (Li, 2017; Poole, 2013; Jing, 2017). Other inflammatory markers may be important in ovarian carcinogenesis. In premenopausal women, ovarian epithelial cells secrete cytokines as part of ovarian function and some of these cytokines are also produced by ovarian cancer cells (Jammal, 2016). Epithelial

cells in proximity to ovulating follicles are likely exposed to these inflammatory mediators that may signal oxidative stress, and enhance the risk of mutagenesis. Importantly, cytokines involved in ovarian function, follicle rupture, and repair (physiologic processes before menopause) are suggested to remain activated in postmenopausal women and may play an etiologic role in ovarian carcinogenesis; these cytokines include: interleukin (IL)-1α, IL-1β, IL-2, IL-6, IL-8, IL-10, tumor necrosis factor alpha (TNF-α), interferon gamma (IFN-γ), granulocyte colony- stimulating factor (G-CSF), and granulocyte macrophage colony-stimulating factor (GMCSF). Many inflammatory mediators, including prostaglandins, leukotrienes, and cytokines, are locally elevated during ovulation. Epithelial cells in proximity to ovulating follicles are likely exposed to these inflammatory mediators that may signal oxidative stress, and enhance the risk of mutagenesis. Moreover, IL-8, an important angiogenesis factor, is elevated in ovarian cancer patients and is believed to be a key factor for cancer growth and new vessel formation (Lane, 2011). Additionally, Saed et al. (2017) has reported that oxidative stress can play an important role in the pathogeneis, neoangiogenesis and dissemination of local or distant ovarian cancer.

Endometriosis is a pelvic disorder associated with inflammation and scarring. Studies also link endometriosis with the increased risk of epithelial ovarian carcinoma through pathways related to oxidative stress and inflammation (Melin, 2006; Worley, 2013). Studies indicate that women with endometriosis differ in the expression of inflammatory mediators, and changes in the cytokine network indicating immune dysregulation, which could contribut to the development of endometriosis (Pizzo, 2002). Wu et al. (2009) performed a study to determine the role of talc in the development of ovarian cancer, considering the history of endometriosis. Results demonstrated an increased risk of ovarian cancer with increasing frequency and duration of talc use; compared to never users, risk was highest among long duration, frequent talc users. A history of physician-diagnosed endometriosis was significantly associated with ovarian cancer in risks, and women who were talc users and had a history of endometriosis showed a 3-fold increased risk, and authors concluded risk of ovarian cancer is significantly associated with talc use and a history of endometriosis.

VIII. MECHANISM OF INFLAMMATION

Inflammation has long been associated with the development of cancer (reviewed by Heidland, 2006; Balkwill, Mantovani, 2001; Rakoff-Nahoum, 2006; Todoric, 2016). An inflammatory process begins when chemical mediators are released by the damaged tissue. The inflammatory response orchestrates host defenses and mediates tissue repair and regeneration in response to damage from chemical toxicants, foreign organisms or carcinogens. Epidemiological evidence points to a connection between inflammation and a predisposition for the development of cancer, i.e., long-term inflammation leads to the development of dysplasia (abnormal cell growth preceding cancer).

Inflammation is a well-established risk factor for all stages of carcinogenesis and tumor progression (Chow, 2012), including ovarian cancer (Maccio and Madeddu, 2012). Inflammation is a factor in a number of mechanisms regarding the etiology of epithelial ovarian cancer and a contributor to

ovarian tumor development and tumor progression (reviewed in Ness, 1999). Inhibition of inflammatory cytokines in the tumor milieu acts on inflammatory-induced angiogenesis and apoptosis and improves prognosis. In a review paper by Ness and Cottreau (1999), talc and asbestos are discussed as risk factors for ovarian cancer, along with endometriosis and pelvic inflammatory disease which are all associated with induction of local cancer.

A. Cytokine Networks

The cytokine networks are very active in producing pro-inflammatory cytokines, growth factors, and chemokines, all of which are molecules active in immune system signalling. There is evidence that inflammatory cytokines and chemokines, which are produced by tumor cells and/or tumor-associated leukocytes, may contribute directly to malignancy. Tumor necrosis factor (TNF)-alpha, a major mediator of inflammation, has actions directed towards both tissue destruction and recovery. TNF can be detected in malignant and/or stromal cells in human ovarian, breast, prostrate, bladder and colorectal cancer, lymphomas and leukemias and often is associated with IL- 1 and -6 and macrophage colony stimulating factor. TNF-α is also implicated in the induction of a chemokine called MCP-1 which can regulate the macrophage and lymphocyte infiltrate and of MMP-9 in the ovarian tumor microenvironment. There is also evidence for pro-cancer actions of TNF-α in animal models. The molecular basis is thought to involve induction of ROS in the form of NO synthase. NO can directly oxidize DNA, resulting in mutagenic changes, and may damage some DNA repair proteins. Inducible NO synthase has been detected in gynegological cancers, including ovarian cancer.

B. Macrophages

The neoplastic process which consists of proliferation, survival and migration is linked with the tumor microenvironment and synchronized with the influx of inflammatory cells, including neutrophils and macrophages which are a main source of exogenous reactive oxygen species (ROS) (Forman and Torres, 2002). Macrophages and the innate immune system can be responsible for tissue injury, when in excess or continuous.

This can also indicate macrophage activation leading to excess production of other macrophage-generated mediators, including cytokines. Macrophages can engulf talc particles and play a critical role in disease. Moreover, macrophages are the major constituents in granulomas. Talc can promote murine macrophage survival and DNA synthesis *in vitro* (Hamilton, 2001). Such enhancement of macrophage survival by talc, if it occurred *in vivo*, could lengthen the cells' tenure in a lesion with the result that more cells would be present to produce inflammatory mediators, such as cytokines, proteinases, and eicosanoids, perhaps potentiated by additional stimuli. This could be another mechanism as to how macrophage cell numbers increase in talc-induced granulomas and inflammatory reactions.

In a 2005 *in vitro* study (Bogatu and Contag, 2005), talc (as a fibrogenic dust) was shown to adsorb high density lipoprotein (HDL). The authors concluded that the adsorption of HDL could have a "causal relationship" with triggering of a fibrotic reaction. The adsorption on the surface of fibrogenic dust particles, including talc provides an opportunity for the intake of HDL by macrophages which then

release an increased amount of fibrogenic mediators. Coating of talc by HDL allows for more rapid uptake by the macrophage as it can use multiple receptors as points of entry into the cell. In general, surfaces of all fibrogenic particles, such as talc, have a specific property which is lacking in non-fibogenic (inert) particles or is at least significantly less effective. However, even upon overloading, non-fibrogenic dusts cannot produce fibrosis.

In another study (Ghio et al., 2012), both mesothelial and airway epithelial cells exposed to talc significantly increased iron importation and concentration of the iron storage protein, ferritin. The production of pro-inflammatory cytokines was also induced by *in vitro* talc exposure relative to control lung tissue, and a time-dependent and concentration-dependent release of oxidents was observed in both cell types. Talc toxicity was also observed in an *in vitro* study comparing effects of micro-scale talc particles with those of smaller nanotalc particles on lung cells (Akhtar, 2010). Cell viability was decreased for all talc exposures, and decreased as a function of talc concentration, origin and particle size. Nanotalc particles differentially induced lipid peroxidation, reactive oxygen species and depletion of the anti-oxidant, glutathione. Further, data suggests that talc toxicity was mediated through oxidative stress.

A study by Khan et al. (2011) demonstrated that nanoscale talc, as opposed to larger talc particles enhanced its cytotoxicity. In this study, macrophages exposed to nanotalc increased the manufacture (transcription) of three macrophage-released pro-inflammatory cytokines and the phosphorylation of two signal transduction pathways. The authors indicated that the inflammatory potential of nano talc particles might be (at least partially) a potential mechanism in talc-mediated pathogenicity.

An early study (Davies et al., 1983) in which the cytotoxicity of seven talcs was evaluated using rat peritoneal macrophage demonstrated modest, but consistent macrophage cytotoxicity visualzed by an increase in macrophage production of two enzymatic cell injury markers including lactate dehydrogenase (LDH) and B-glucuronidase (compared to *in vitro* treatment with a non-fibrogenic dust. This study points to the potential of talc to “activate” macrophage leading to increased production of macrophage-released mediators including pro-inflammatory cytokines. Some investigators have suggested such *in vitro* macrophage changes could predict fibrogenicity *in vivo*. Based on talc chemical analyses, the authors concluded that effects on macrophages were not due to contaminating minerals.

In a molecular cell study by Shukla et al. (2009), non-fibrous-containing talc at low concentrations caused increased expression of the gene Activating Transcription Factor (ATF genes modulates production of pro-inflammatory cytokines and growth factors in human lung cells) in cultured mesothelial cells at 8 hr and no changes at 24 hr, whereas expression levels of 30 genes were elevated at 8 hr at high talc concentrations.

Tumor necrosis factor (TNF)-α is a cell signaling protein produced by macrophages, primarily involved in the regulation of immune cells. Pre-diagnostic serum levels of 46-inflammation –related biomarkers were measured in 149 incident ovarian cancer cases and matched controls. As his been discussed in several aforementioned sections of this Report, C-reactive protein (CRP), IL-1-α and TNF-α proved to all be significantly elevated and associated with increased cancer risk. In analyses restricted to serous ovarian cancer (n=83), the associations with CRP and IL-8 remained or strengthened. Thus, IL-8

can also be considered an inflammatory biomarker of ovarian cancer (Trabert et al., 2014), again demonstrating talc's action as an inflammatory agent. Iron and its homeostasis are intimately tied to the inflammatory response (Wessling-Resnik, 2010). Talc has been shown to modulate TNF- α and IL-6 production by its binding to iron (Ghio, 2011). TNF-α, like CRP, is a marker of various inflammation processes. TNF-α has been shown to play a role in later steps of carcinogenesis. For example, NF-κB activation by TNF- α is involved in neoplastic transformation, proliferation, and tumor survival. In addition, in ovarian cancer cells, TNF-α enhances cell migration and metastasis through the action of NF-κB. TNF-α was positively associated with ovarian cancer in case-control studies using serum samples collected at diagnosis.

C. Role of Oxidants in Ovarian Cancer

The chronic inflammatory states associated with infection and irritation may lead to environments that foster genomic lesions and tumor initiation. One effector mechanism by which the host system responds to insult is production of free radicals such as reactive oxygen species (ROS), hydroxyl radical (OH•) and superoxide (O_2-•) and reactive nitrogen species (RNS), nitric oxide (NO•) and peroxynitrite (ONOO). Primarily thought to be anti-microbial, these molecules form due to the activities of host enzymes such as myeloperoxidase, NADPH oxidase, and nitric oxide, which are regulated by inflammatory signaling pathways. Importantly, ROS and RNS lead to oxidative damage and nitration of DNA bases which increase the risk of DNA mutations.

During inflammation, macrophages, mast cells and neutrophils are recruited to the site of damage, which leads to a 'respiratory burst' due to an increased uptake of oxygen, and thus, an increased release and accumulation of ROS at the site of damage. A sustained inflammatory/oxidative environment leads to a vicious circle, which can damage healthy neighboring epithelial and stromal cells and over a long period of time may lead to carcinogenesis. Oxidative stress can also activate a variety of transcription factors. Activation of these transcription factors can lead to the expression of over 500 different genes, including those for growth factors, inflammatory cytokines, chemokines, cell cycle regulatory molecules, and anti-inflammatory molecules that can also be linked to cancer. Under a sustained environmental stress, ROS are produced over a long time, and thus significant damage may occur to cell structure and functions that could induce neoplastic transformation. In general, the longer the inflammation persists, the higher the risk of cancer.

Following an inflammatory stimulus, initiation of carcinogenesis mediated by ROS may be direct (oxidation, nitration, halogenation of nuclear DNA, RNA, and lipids), or mediated by the signaling pathways activated by ROS (Reuter, 2010; Saed, 2011; Saed, 2017). Hydrogen peroxide plays an important role in carcinogenesis because it is capable of diffusing through cell membranes and producing many types of cell injury. NO is another free radical implicated in carcinogenesis (Saed, 2017). iNOS, calcium-independent isoform, produces large amounts of NO and is only expressed during inflammation. ROS can specifically activate certain signaling pathways and thus contribute to tumor development through the regulation of cellular proliferation, angiogenesis, and metastasis.

1. Talc-Induced Inflammation and Oxidative Stress

Even a single dose of a carcinogen can produce effects that are adverse to cells and tissue at the site of exposure. *In vitro* studies provide a safe and effective vehicle by which to measure those effects in a controlled environment.

Carcinogenic potential of any compound can be determined by performing a well-established methodology called a neoplastic cell transformation assay. In a 2007 study by Buz'Zard, two human ovarian cell culture lines were treated in vitro with talc from 24 to 120 hr (Buz'Zard, 2007). Another group of talc-treated cells were also treated with a specific anti-inflammatory inhibitor to determine whether talc produced transformation through the production of inflammation. Following talc treatment of both ovarian cell types, the cells' ability to grow in suspension, a key characteristic of neoplastically transformed cells, was measured - non-neoplastically-transformed normal cells cannot grow in suspension. Results showed that treatment with talc can transform ovarian cells which further demonstrates the carcinogenic potential of talc. As anti-inflammatory treatment reduced formation of ROS and number of transformed colonies, a relationship between cell transformation and inflammation was demonstrated. Interestingly, exposure of ovarian cells to talc also increased ROS generation in this study in a time and dose-dependent manner. These effects could be linked with neoplastic changes as chronic inflammation is associated with cancer induction and ROS are often seen as a component of the tumor microenvironment. Human neutrophils exposed to talc in this study also increased ROS generation significantly compared to control phagocytes.

In a study carried out by Keskin in 2009, rats exposed to talc produced an increase in ovarian follicles which could be related to the " ovulation theory" associated with ovarian cancer, thus demonstrating a plausible mechanism for talcum powder-induced ovarian cancer.

Recent data demonstrates the importance of oxidative stress in ovarian cancer. The effects of talcum powder exposure on oxidative stress levels in normal ovarian epithelial cells, ovarian epithelial cells and cancerous ovarian epithelial cells were measured (Saed, 2017; Fletcher, 2018 (abstract)). Studies indicate that epithelial ovarian cancer manifests a persistent pro-oxidant state through alteration of the redox balance by the up-regulation of several oxidant enzymes in epithelial ovarian caner tissues (Saed, 2018). Advancing similar work, in a recently accepted abstract, Harper and Saed report a mechanism by which talc enhances the pro-oxidant state in normal (ovarian and tubal) and ovarian cancer cells, through induction of gene point mutations (corresponding to known specific single nucleotide polymorphisms - SNPs) in key oxidant enzymes, altering their activities (Harper and Saed, 2018).

Emerging science by Fletcher (2018) demonstrated that talc-treated ovarian cancer cell lines and normal ovarian epithelial cells showed a marked increase in mRNA levels of pro-oxidant enzymes, including iNOS and MPO. This shift to a pro-oxidant environment indicates oxidative stress as early as 24 hours after exposure. These recent facts provide strong support for the ability of talc to produce an oxidant state that leads to inflammation and in turn epithelial ovarian cancer. This latter study shows that talcum powder enhances the redox state as part of the inflammatory cascade in both normal ovarian

epithelial cells and in ovarian cancer cells, revealing a plausible mechanistic underpinning for talc-induced ovarian cancer.

Another study by the same authors showed that talcum powder exposure increased levels of the cancer antigen, CA-125, in both normal ovarian cells and ovarian cancer cells. (Fletcher and Saed, 2018). CA-125 is an antigen that is elevated in some patients with specific types of cancers, and is used as a biomarker for ovarian cancer detection, providing further information about talcum powder's carcinogenic properties.

In a study by Shim et al. (2015), inhalation of talc revealed infiltration of macrophages and the increased expression of the antioxidant, superoxide dismutase indicating oxidative stress in rats. Moreover, in the same study inhalation of talc demonstrated macrophage aggregations and oxidative damage in the lungs. Intrapleural injection of talc particles produced an acute serum inflammatory response, more pronounced with smaller particles (Genofre et al., 2009). In addition, talc exposure induced vasoconstriction in the brain via the action of superoxide anions (Mori et al., 1995). Non-fibrous talc at low *in vitro* exposure concentrations caused increased expression of transcription factors associated with the inflammatory process in a time and dose-dependent manner (Shukla et al., 2009). Nano-talc exposure enhanced the production of pro-inflammatory cytokines by macrophages *in vitro* (Khan et al., 2011). Also, pre-treatment of macrophage (prior to talc exposure) with inflammatory signal transduction inhibitors reduced TNF mRNA stability demonstrating their role in TNF mRNA stabilization and expression (Khan et al., 2011).

In an epidemiological study, talc exposure was significantly associated with ovarian cancer in women who lacked a specific anti-oxidant genotype (glutathione-S transferase M1/T1) (Gates et al., 2008). Finally, talc exposure increases COX2, an enzyme that plays a critical role in inflammation (Pace et al., 2006).

At high concentrations or chronic exposure, ROS can damage cellular macromolecules and contribute to neoplastic transformation and/or tumor growth. Other likely manifestations of talc-induced inflammation include reduced fibrinolysis, activation of neutrophils and macrophages and increased production of cytokines and growth factors, and these have been suggested to occur in the peritoneum in response to contamination by surgical glove powder (Merritt et al., 2008).

In sum, inflammation is a primary mediator of ovarian cancer. As the scientific studies outlined above demonstrate, talcum powder products cause inflammation that can result in an elevation of biomarkers; changes in cell signaling; activation of chemokines and cytokines; changes in the oxidative environment; gene alterations and/or mutations; inhibition of apoptosis and induces neoplastic transformation and prolifearation (i.e., cancer). This talcum powder-induced inflammatory cascade provides significant biologic and toxicologic support for a conclusion that talcum powder products can cause ovarian cancer.

D. Iron-Facilitated Inflammation

Talc particles can bind iron and iron facilitates inflammation and ROS production; surfaces of silicates including talc has a net negative charge on the surface which generates a capacity for the adsorption and exchange of cations like iron which has a high affinity for oxygen-donor ligands. According to J&J documents from Luzenac America Technical Center, heavy metal analyses on Grade 66 Non-Shear Disk Test Run samples demonstrated very high levels of iron (15,200 – 21,500 mg/kg) that could cause oxidative stress and an inflammatory response. Multiple studies have demonstrated that exposure to talc disrupts iron homeostasis, oxidative stress, and causes a fibro-inflammatory response (Akhtar et al., 2010; Ghio et al., 1992; Ghio et al., 2012). Talc exposure significantly increases iron importation and concentrations of ferritin (iron storage protein). The accumulation of iron, the accompanying oxidative stress, and inflammatory events after exposure to talc are comparable to those with other forms of particulate matter. The capacity of talc particles to support the *in vitro* generation of oxidants in an acellular environment was significantly affected by the concentration of associated iron, with talc-Fe producing a significantly greater signal for lipid peroxidation relative to talc alone (Akhtar, 2010). This relationship is supported by inhibition of the effect by addition of a metal chelator and a hydroxyl radical scavenger. The disruption of cell iron homeostasis is frequently associated with oxidative stress and inflammation.

IX. SUMMARY OF OPINIONS

I hold the following opinions to a reasonable degree of scientific certainty:

1. Based on the scientific literature and the testing results that I have seen by Defendants and Drs. Longo and Rigler, it is my opinion that talcum powder products, including Johnson's Baby Powder and Shower to Shower, may contain known carcinogens, including asbestos, fibrous talc, and heavy metals. In addition, these products contain fragrance chemicals, many of which are inflammatory agents, toxicants, or potential carcinogens.

2. Talcum powder can reach the ovaries through two routes with anticipated use: 1) perineal application (dermal) with migration/transport through the genital tract via the vagina, uterous, and fallopian tubes; and, 2) inhalation of talcum powder particles. Through either route, talcum powder and its constituents could reach the lymphatic system and bloodstream.

3. Exposure to talcum powder products causes an inflammatory tissue reaction which may result in the following:
    a. Elevation of increased inflammatory markers;
    b. Changes in cell signaling;
    c. Activation and/or release of chemokines and cytokines;
    d. Changes in the oxidative environment;
    e. Gene alterations and/or mutations;
    f. Inhibition of apoptosis; and

g. Neoplastic transformation and proliferation

4. Based on knowledge of the carcinogenic components of talcum powder products, the potential of the powder, with its components, to reach the ovaries and the resultant inflammatory tissue response, it is biologically plausible for talcum powder products to cause ovarian cancer.

I reserve the right to amend or modify this report as new information becomes available. I have not testified in litigation over the previous 4 years. I am charging $ 350 per hour for my work on this matter.

Exhibit A

JUDITH TERRY ZELIKOFF, Ph.D.
Tenured Professor

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EDUCATION

1973: Bachelor of Science (**Biology**)
Upsala College
East Orange, NJ

1976: Master of Science **(Microbiology)**
Farleigh Dickinson University
Department of Biology
Teaneck, NJ,
in conjunction with,
UMDNJ-New Jersey Medical School
Department of Neuroscience
Newark, NJ
Thesis Dissertation: Herpes Simplex Virus-IgM Specific Antibodies in Guillian-Barre Syndrome

1982: Doctor of Philosophy (**Experimental Pathology**)
UMDNJ-New Jersey Medical School
Department of Pathology
Newark, NJ
Thesis Dissertation: Cytoskeletal Modifications of Human Fibroblasts that Occur During a Complement-Dependent Cytotoxic Antibody Response

PROFESSIONAL EXPERIENCE

1982-Present: ***NEW YORK UNIVERSITY SCHOOL OF MEDICINE***
Institute of Environmental Medicine
Tuxedo, NY

2005- Present: Tenured Professor
Laboratory of Pulmonary & Systemic Toxicology

Developmental Immunotoxicology: Effects of fetal insults on later life immune-related diseases in the offspring.

Pulmonary Immunotoxicology: Characterization of inhaled metal, gaseous, and airborne pollutant mixtures including woodsmoke and tobacco smoke, on pulmonary immune defense mechanisms and host resistance against infectious disease and asthma.

Environmental Toxicology/Ecoimmunotoxicology: Effects of aquatic pollutants on the immune responses of fish; development of immune biomarkers. Alternate animal models for immunotoxicological studies.

1995-2005: Associate Professor (Tenured in 1997)
Laboratory of Systemic Toxicology

1989-1995: Assistant Professor
1986-1989: Research Assistant Professor
Laboratory of Pulmonary Biology
Laboratory of Environmental Toxicology

Environmental Toxicology: Characterization of aquatic pollutants and immune defense mechanisms of fish. Studies concerning drug bioaccumulation and metabolism in different fish species.

Inhalation/Pulmonary Toxicology: Effects of ambient pollutants on macrophage metabolism and immune function.

1984-1986: Associate Research Scientist
Laboratory of Environmental Toxicology

Genetic Toxicology: Clastogenic/mutagenic effects of complex environmental mixtures.

Cell Biology: Establishment of primary cultures for assessing the toxicity of environmental contaminants *in vitro*.

1982-1984: NIH (NHLBI) Post-Doctoral Fellow
Laboratory of Environmental Toxicology

Genetic Toxicology: Development of short-term *in vitro* bioassays to detect carcinogens, promoters and co-carcinogens in complex environmental mixtures.

1977-1978: *PFIZER PHARMACEUTICAL*
Laboratory of Chemical Carcinogenesis
Maywood, NJ

Assistant Research Scientist
Laboratory studies using animal models and *in vitro* mammalian cell systems to investigate chemical- and viral-induced carcinogenesis.

1974-1975: *VA HOSPITAL /UMDNJ-NEW JERSEY MEDICAL SCHOOL*
Department of Neuroimmunology
East Orange, NJ

Associate Research Scientist
Laboratory studies investigating the etiology of viral-induced neuropathologies

TEACHING EXPERIENCE - NATIONAL

1990-Present: *NEW YORK UNIVERSITY SCHOOL OF MEDICINE*
Department of Environmental Medicine
Tuxedo, NY
Graduate Courses

- Global toxicology & community health (NYU Global College of Public Health: Organizer/Director, Fall, 2018; offered every year)
- Environmental Immunotoxicology (Organizer/Director, 1993-present)
- Organ System Toxicology (Director, 2001-present)
- Toxicology (Biology-cross linked: Director, 2010 – present)
- Communication Skills (Lecturer; 2010-present)
- Principles of Toxicology (Lecturer; 1992-present)
- Environmental Physiology of the Respiratory Tract (Lecturer; 1992– 1994)

1979-1994: *WILLIAM PATERSON COLLEGE*
Department of Biology
Wayne, NJ

Adjunct Professor
Undergraduate Courses

- Microbiology lecture and laboratory (1979 - 1984)
- Human biology lecture and laboratory (1979 - 1994)

1991-1994: *ROCKLAND COMMUNITY COLLEGE*
Department of Biology
Suffern, NY

Adjunct Professor
Undergraduate Courses

- Microbiology lecture and laboratory

1979-1982: *SETON HALL UNIVERSITY*
Department of Biology
South Orange, NJ

Research Scientist/Graduate Assistant
-Laboratory studies in immunopathology, virology, viral immunology, and microbiology

-**Undergraduate and Graduate Courses**

- Bacteriology lecture and laboratory
- Advanced Microbiology
- Cell biology/Virology techniques

1976-1979: *FAIRLEIGH DICKINSON UNIVERSITY*
Department of Biology
Teaneck, NJ

Adjunct Professor
Undergraduate and Graduate Courses

- General biology lecture and laboratory

- Human genetics
- Immunology

TEACHING EXPERIENCE - INTERNATIONAL

2013-present *UNIVERSITY OF PORT HARCOURT (Port Harcourt, Nigeria)*
Dept. of Toxicology
Lecturer in graduate toxicology course

2002-present: *CHULABHORN RESEARCH & GRADUATE INSTITUTE (Professor, Course Director)*
Department of Toxicology
Bangkok, Thailand
Graduate Course (3 weeks- given every even year)
- Environmental Immunotoxicology and Reprotoxicology

1999

1999-2000: *UNIVERSITY OF TASMANIA (Adjunct Professor)*
Department of Environmental Toxicology
Tasmania, Australia
Graduate Course (2 weeks)
- Fish Immunology & Immunotoxicology (Organizer/Director; Lecture and Lab)

1999-2000: *LINCOLN UNIVERSITY*
Department of Environmental Health Sciences
Christ Church, New Zealand
Graduate Course (2 weeks)
- Fish Immunology & Immunotoxicology (Organizer/Director; Lecture and Lab)

HONORS AND AWARDS

- 2018 – Society of Toxicology (SOT), Education Award
- 2015 – SOT, Women in Toxicology Mentorship Award
- 2013 – West African SOT (WASOT), Distinguished Recognition
- 2012 - 2014, SOT, Distinguished Service as SOT Secretary
- 2012 - SOT, Global Senior Scholar Host Award
- 2012 – SOT, Career Achievement Award in Immunotoxicology
- 2008 – Mid-Atlantic Chapter Society of Toxicology, President

PUBLICATIONS

Peer-reviewed Journals (In ascending order)

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2. **Zelikoff, J.T**., J.M. Daisey, K. Traul and T.J. Kneip. Balb/c 3T3 cell transformation response to organic extracts of airborne particulate matter as seen by their survival in aggregate form. *Mutat. Res.* 144: 107-116 (1985).

3. **Zelikoff, J.T**., N. Atkins, T.G. Rossman and J.M. Daisey. Cytotoxicity of fine particles with and without absorbed polycyclic aromatic hydrocarbons using Chinese hamster lung cells (V79). *Environ. Internat.* 11: 331-339 (1985).

4. **Zelikoff, J.T**., N. Atkins and S. Belman. Stimulation of cell growth and proliferation in NIH-3T3 cells using onion and garlic oil. *Cell Biol. Toxicol.* 2: 369-378 (1986).

5. Ende, J., J. Grizzanti, E.V. Orsi, P.P. Lubanski, R.C. Amarusso, L.B. Reichman and **J.T. Zelikoff.** Sarcoid and cytotoxic lung antibodies. *Life Sciences* 39: 2435-2440 (1986).

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8. **Zelikoff, J.T**., J.H. Li, A. Hartwig and T.G. Rossman. Genetic toxicology of lead compounds. *Carcinogenesis* 9: 1727-1732 (1988).

9. Schlesinger, R.B., A.F. Gunnison and **J.T. Zelikoff.** Modulation of pulmonary eicosanoid biosynthesis following exposure to sulfuric acid. *Fundam. Appl. Toxicol.* 15: 151-162 (1990).

10. Schlesinger, R.B., K.E. Driscoll, A.F. Gunnison and **J.T. Zelikoff.** Pulmonary arachadonic acid metabolism following acute exposures to ozone and nitrogen dioxide. *J. Toxicol. Environ. Health* 31: 275-290 (1990).

11. Schlesinger, R.B., L.C. Chen and **J.T. Zelikoff.** Comparative potency of inhaled acidic sulfate aerosols: The influence of specific components and the role of H^+ ions. *Environ. Res.* 52: 210-224 (1990).

12. Schlesinger, R.B., P.A. Weideman and **J.T. Zelikoff.** Effects of repeated exposure to ozone on respiratory tract prostanoids. *Inhal. Toxicol.* 3: 27-36 (1991).

13. **Zelikoff, J.T.**, N.A. Enane, D. Bowser, K.S. Squibb and K. Frenkel. Development of fish peritoneal macrophages as a model for higher vertebrates in immunotoxicological studies. I. Characterization of trout macrophage morphological, functional and biochemical properties. *Fundam. Appl. Toxicol.* 16: 576-589 (1991).

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15. Costa, M., N.T. Christie, O. Cantoni, **J.T. Zelikoff**, X.W. Wang and T.G. Rossman. DNA damage by mercury compounds: An overview. Proc. of Advances for Mercury Toxicology. In *Advances in Mercury Toxicology* (T. Suzuki, Ed.), Plenum Press, NY. pp. 255-273 (1991).

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17. **Zelikoff, J.T**. and R.B. Schlesinger. Immunomodulation by sulfuric acid aerosol: Effects on pulmonary macrophage-derived tumor necrosis factor and superoxide production. *Toxicology* 76: 271-281 (1992).

18. Cohen, M.D., E. Parsons, R.B. Schlesinger and **J.T. Zelikoff.** Immunotoxicity of *in vitro* vanadium exposure: Effects on interleukin-1, tumor necrosis factor, and prostaglandin E2 production by macrophages. *Int. J. Immunopharmacol. Immunotoxicol*. 15: 437-446 (1993).

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26. Ng, S.P**., Zelikoff J.T.** Tumor challenges in immunotoxicity testing. Vol. 599. Humana Press, Springer Science. Immunotoxicity Testing: Methods and Protocols, Methods in Molecular Biology. (2018)
27. **Zelikoff, J.T.**, and M.D. Cohen. Pulmonary Immunology. In: *Comprehensive Toxicology*. (C. McQueen, Ed.). Elsevier, UK. 2018.

INVITED NATIONAL AND INTERNATIONAL LECTURES/PRESENTATIONS (Present – 2000, *in descending order***):**

August 2018: International Society of Exposure Science (ISES); International Society for Environmental Exposure (ISEE). *Contamination of the Ramapough Nation: A toxic legacy.* **Environmental contamination and Indigenous populations symposia.** Ontario, Canada.

February 2018: Louisiana State University. Electronic cigarettes and pregnancy: Lessons learned from mice. Baton Rouge, LA

January 2018: Mt. Holyoke College. What's safer for the unborn child: electronic cigarettes or air pollution? MA.

December 2017: Texas A & M. Prenatal exposure to ambient particulate matter impacts cardiovascular development. TX.

December 2017: International Conference on Environmental Impacts. Air pollution and pregnancy. Deradun, India

November 2017: International Conference on "Impact of Environment on Women's Health: **Amity University Uttar Pradesh**. Maternal exposure to particulate air pollution during pregnancy and Impacts on fetal health: What are we learning from animal studies? Lucknow, India.

November 2017: **American Public Health Assoc. (APHA) Annual Meeting**. Identifying Environmental concerns, environmental exposures and health concerns in the Ramapough Lenape Tribe. Atlanta, GA.

October 2017: International Society of Exposure Science. A community in toxic crisis: Ramapough Native Americans. Durham, NC.

April 2017: Queensborough College. Neurocognitive effects of E-cigarettes. Queens, NY.
July 2016: NIOSH seminar. Reproductive implications of Nanomaterials. WV
July 2016: EPA seminar. Ambient particulate matter and cardiotoxicity. Chapel Hill, NC.
June 2016: Workshop on Nanomaterials and the fetal-placental unit. Prenatal Nephrotoxicity and Maternal Nanomaterial Inhalation. Boston, MA.
May 2016: NIH Tobacco Research. Toxicological assessment of smokeless tobacco products: A systematic ranking system. Bethesda, MD
April 2016: AHA, ATrac Meeting. Toxicity ranking of alternative tobacco products. Louisville, KY.
March 2016: Society of Toxicology: Course in Medical Education. Effects of fracking on reproductive and developmental health. New Orleans, LA
March 2016: Society of Toxicology: Symposia on Fracking and Health. Effects of fracking on reproductive and developmental health. New Orleans, LA
February 2016: American Association for Advancement of Science: Symposia on Alternative Tobacco Products and Health. Early life exposure to alternative tobacco products as a major risk factor of later life chronic disease. Washington, DC
October 2015: 7th International Symposia on Nanotechnology and Occupational and Environmental Health. Reproductive and developmental toxicity of gold nanoparticles in a mouse model of pulmonary exposure. Limpopo Province, South Africa.
May 2015: Amer. Assoc. Immunol. Maternal inhalation of ambient particulate matter causes alterations in immune profiles and anti-tumor mechanisms in juvenile murine offspring. New Orleans, LA.
April 2015: Wayne State University, *CURES Seminar Series at Wayne State University's Institute of Environmental Health Sciences*. Maternal exposure to particulate air pollution during pregnancy impacts fetal development and neonatal growth in a mouse model.
March 2015: **Society of Toxicology**. Symposia on: New and Emerging Tobacco Products—Biomarkers of Exposure and Injury (Chair). Reproductive/Developmental effects of exposure to new and emerging tobacco products and to nicotine delivery devices in a mouse model. San Diego, CA.
Dec. 2014: University of Illinois – Maternal exposure to ambient particulate matter during particular gestational windows produce developmental and reproductive consequences in a mouse model. Urbane, IL.
July 2014: Oregon State University – Early life nanoparticle exposure brings early and later life health consequences. Corvallis, OR.
March 2014: Society of Toxicology – Tobacco products and prenatal exposures. Phoenix, Arizona.
February 2014: ***West African Society of Toxicology*** – Air pollution in developing nations. Lagos, Nigeria.
January 2014: ***Ernst Strungmann (ES) Forum, (Rapporteur)-*** *Heavy metals and infectious disease.* Frankfurt Germany.
November 2013: American Chemical Council. Risk Assessment and Communication, Working Group. Washington, DC.
October 2013: ***First International Conference on Waterpipe Tobacco Research.*** Working Discussion Group Leader: Abu Dhabi.
October 2013: ***NIH-sponsored Workshop in South Asian Diversity Populations and Health Effects.*** Sloan Kettering Cancer Center. Working Group member on smokeless tobacco. NY, NY.

June 2013: ***FDA, Center for Tobacco Control***. Public health impacts of fetal exposures to tobacco & environmental toxicants: From early life to adult disease and policy needs. MD

March 2013: ***Society of Toxicology, Committee on Diversity Initiatives*** – Exposure to smoked and smokeless tobacco *in utero*: Fetal injury and life long consequences. San Antonio, TX

February 2013: ***Nigeria University*** – Smokeless tobacco: A global look at the problem, Port Harcourt, NIGERIA

February 2013: ***FDA: Center for Medical Devices*** – Fetal basis of adult disease: early life exposure to environmental and occupational toxicants. Silver Spring, MD.

October 2012: ***Memorial Sloan Kettering*** – Arsenic contamination in Bangledesh. New York, NY

May 2012: ***Memorial Sloan Kettering*** – Toxicology of Smokeless tobacco. NY, NY.

April 2012: ***University of Connecticut*** – Tobacco products *in utero* are associated with later life disease outcomes. Storrs, CT.

March 2012: ***Biomass Symposium*** – Toxicological implications for domestic burning. Feb. 2012: **NYU Medical Center, Dept. of Psychiatry** - Chemical stressors *in utero* and later life disease outcomes. New York, NY.

Jan 2012: *British American Tobacco –* *In vitro* translational studies and the toxicology of smoking. Southampton, UK.

Dec. 2011: FDA – ***The reproductive effects of cadmium nanoparticles***. Reston, VA.

Dec. 2011: **NYU Dept. of Bioethics** – Cigarette smoking & smokeless tobacco: Is there really a good choice? New York

Oct. 2011: NorCal SOT **– Fetal basis of adult disease – the role of maternal smoking**. Menlo Park, CA.

Sept. 2011: ***European Aerosol Conference – Plenary Lecture*****:** The toxicology of biomass combustion emissions. Satellite Workshop on Biomass Combustion, Manchester, England.

March 2011: ***NYU Ethics Forum*** - Exposure to Cigarette Smoke *in Utero*: Fetal injury and Life Long Consequences**.** New York

March 2011: *NYU Medical Center, Dept. of Obstetrics and Gynecology Grand Rounds* – Early life insult by tobacco smoke and later life disease susceptibilities. March 15, 2011

March 2011: *Society of Toxicology, Committee for Diversity Interests* – Cigarette exposure *in utero*: You are what you breathe. Washington, DC. March, 2011.

Nov. 2010: *Texas A & M University –* Early life exposure to cigarette smoke suppresses anti-tumor immune defenses of the prenatally exposed offspring in a mouse model" College Station, TX.

May 2010: ***Workshop on Emissions and Health Impacts of Biomass Fuels*** – Health effects of woodsmoke: A toxicological model for mechanisms and policy needs. Penn State, State College, PA.

March 2010: ***Environmental and Occupational Health Sciences Institute, Rutgers University*** - Fetal exposure to cigarette smoke mediates anti-tumor immune mechanisms in adult murine offspring. New Brunswick, NJ. March, 2010.

March 2010: ***Society of Toxciology, Committee for Diversity Interests –*** Exposure to cigarette smoke in utero: Fetal injury and life-long consequences. Salt Lake City, UT.

Nov. 2009: *United Nations Environmental Programme –* Toxicological assessment of the atmospheric brown cloud. Incheon, Korea.

Sept. 2009: *7th Congress of Toxicology in Developing Countries* – Fetal insult and later onset diseases. Sun City, South Africa.

August 2009:***Japanese Society of Immunotoxicology*** – Prenatal exposure to cigarette smoke increases tumor susceptibility of juvenile mice via changes in anti-tumor immune mechanisms. Asahikawa, Japan.

May 2009: ***Asia-Pacific Forum on Andrology***, Hormonal changes accompanying cigarette smoke induced preterm births in a mouse model. Nanjing China.

Dec. 2008: ***St. Johns University*** – Mechanistic insights into offspring cancer risk associated with maternal smoking. Queens, NY.

August 2008: ***U.S. EPA, National Center for Environmental Assessment*** - Gender-related effects on offspring tumor risk and response to prenatal cigarette smoke exposure may be related to testosterone: a toxicological model. Washington, DC.

June 2008: ***Institute for Science and Health (IFSH)* –** Early exposure to cigarette smoke may serve as an indicator of chronic diseases in the offspring later in life. Cardiff, Wales.

March 2008: ***Society of Toxicology* –**Prenatal exposure to tobacco smoke induces asthma-related responses in non-sensitized female offspring later in life. Seattle, Washington.

March 2008: ***Society of Toxicology*** – Prenatal exposure to cigarette smoke: Are our children paying the price? Seattle, Washington. March 2008.

August 2007: ***United Nations Environmental Program (UNEP)*** – Toxicology of the Atmospheric Brown Cloud (ABC). Seoul, Korea.

March 2007: ***University of Louisville (KY)*** – Increased cancer risk: A possible birth defect associated with maternal smoking. Louisville, KY.

March 2007: ***Institute for Science and Health (IFSH)*** – Prenatal cigarette smoke exposure and offspring asthma. Louisville, KY.

Feb. 2007: ***International Conference on Environment: Survival and Sustainability* -** Sustaining a healthy fetal environment: A little told threat of increased cancer and asthma risk for the juvenile offspring exposed prenatally to cigarette Smoke. Near East University, Nicosia-Northern Cyprus.

Feb. 2007: ***International Conference on Environment: Survival and Sustainability*** - Contamination of aquatic environments with polychlorinated biphenyls (PCBs) or benzo(a)pyrene (B[a]P) can adversely impact the immune health and sustainability of inhabiting Fish. Near East University, Nicosia-Northern Cyprus.

Dec. 2006: ***Philip Morris External Review Symposia* –** Effects of prenatal exposure to cigarette smoke on tumor development and immune surveillance mechanisms in the developing offspring: A toxicological model. Landsdowne, VA. Dec. 2006.

May 2006: ***MidAtlantic Chapter of Society of Toxicology (MASOT)*** – Increased cancer risk in the offspring: A birth defect associated with maternal smoking. Scotch Plans, NJ.

April 2006: ***University of Guelph*** – Maternal smoking and cancer: Are the unborn children paying the price? Kempville, Ontario Canada.

March 2006*: Institute for Science and Health* – Prenatal exposure to mainstream cigarette smoke alters susceptibility of the offspring to asthma. Vienna, Austria.

March 2006: ***Society of Toxicology* –** Maternal smoking and cancer: Are the unborn children paying the price? San Diego, CA.

October 2005: ***Chulabhorn Research Institute*** – *Immunotoxicology: A new focus for Thai science.* Scientific Research Institute of Thailand. Bangkok, Thailand.

May 2005: ***American Thorasic Society* -** Immunotoxicological mechanisms of prenatally-exposed respiratory contaminants. Symposia on "Impact of prenatal and early infancy environmental exposures on neonatal and infant health". San Diego, CA..

May 2005: ***California Society of Environmental Toxicology and Chemistry –*** Mechanisms of Fish Immunotoxicity. Berkley, CA.
April 2005: ***Life Science Research Organization (LSRO)*** **–** Prenatal exposure to cigarette smoke increases tumor susceptibility in the offspring: A toxicological model. St. Louis, MO.
March 2005 - ***Society of Toxicology*** – Immunotoxicity of prenatal mainstream cigarette smoke exposure. Symposia on "Mechanisms Linking the Lung and Immune System". New Orleans, LA.
Feb. 2005: ***Institute for Science and Health (IFSH)*** – Effects of in utero cigarette smoke exposure on asthma development in the offspring. Washington, DC.
Feb. 2005: ***Canadian Lung Association*** – Health Effects of Woodburning. New Brunswick, Canada.
Nov. 2004: ***Environmental Mercury Research Forum*****.** Metal toxicity in aquatic organisms. Energy & Environmental Research Center (U. of North Dakota). Grand Forks, ND.
Oct. 2004: ***VIIth Annual Conference of Soil, Sediments and Water*****.** Immunological Alterations as Bioindicators of Environmental Health. Amherst, MA.
Sept. 2004: ***Slovenian Society of Toxicology*** – Immunological biomarkers. Lublijana, Slovenia.
March 2004: ***Society of Toxicology*** **–** Inhalation of concentrated ambient particulate matter and associated metals increases host susceptibility to pulmonary pneumonia. Baltimore, MD.
Jan. 2004: ***University of Arizona*** **–** Toxicological impact of inhaled wood smoke on pulmonary antimicrobial defense. Tucson, AZ.
Jan. 2004: ***College of Staten Island*** **–** Toxic insult and human health effects: Lessons learned from an aquatic species. Staten Island, NY.
Dec. 2003: Sixth National Environmental Public Health Conference (Center for Disease Control) Woodsmoke: A closer look at public health concerns and mechanisms of toxicity. Atlanta, GA.
Nov. 2003: ***Society of Environmental Toxicology and Chemistry*** **-** Immunotoxicology and Risk Assessment. Austin, TX.
Oct. 2003: ***Chulabhorn Research Institute*** **–** Immunotoxicology Course Series (10d). Bangkok, Thailand.
June 2003: ***International Symposium on Pharmaceutical Sciences*** **-** Health Effects of Inhaled Particulates. University of Pharmaceutical Sciences. Ankara, Turkey.
June 2003: United States Army Center for Environmental Health Research - Immune Assays for Hazard Assessment and Species Extrapolation. Fort Detrick, MD.
May 2003 - ***Pollutant Responses of Marine Organisms (PRIMO)*** **-** Immunotoxicology in Fish. Tampa, FL.
March 2003: ***Society of Toxicology*** - Woodsmoke: Cozy Atmosphere or Public Menace? Salt Lake City, UT.
Nov. 2002: ***Society of Toxicology and Chemistry*** **-** Immune Biomarkers for Use in Ecological Risk Assessment. Salt Lake City, UT.
Oct. 2002: ***Padova University*** **-** Lessons Learned About Human Health From Aquatic Species. Padova, Italy.
Oct. 2002 - ***Slovenia Society of Toxicology*** **-** Biomarkers for Ecotoxicology. Ljubljana, Slovinia.
Sept. 2002: ***University of Florida*** **-** Effects and Mechanisms of Benzo(a)pyrene-induced Immunosuppression in Fish. Gainsville, FL.

June 2002: ***Yale University, Dept. of Occupational and Environmental Medicine* -** Lessons on Human Health and Toxic Impact Learned from our Aquatic Counterparts.

Sept. 2001: *Third International Meeting on Molecular Mechanisms of Metal Toxicity and Carcinogenicity* - Immunodysfunction: An underlying Mechanism of Metal Toxicity in Aquatic Organisms. Sardinia, Italy.

July 2001: ***Pollutant Responses in Marine Organisms* -** Immunotoxicology in fish - Applications and Mechanisms of Response. Plymouth, England.

Oct. 2000: *Conference on Women in Science* - Aging: Good or Bad News for the Immune Response. Rutgers University. New Brunswick, NJ.

Oct. 2000: *International Conference on Environmental and Occupational Lung Disease* - Woodsmoke Impairs Host Resistance Against Pulmonary Infections in an Animal Model. Lucknow, India.

May 2000: *EPA-Duluth* - Fish Immune Status: A Sensitive System for Assessing Toxicological Impact of Aquatic Environments. Duluth, MN.

May 2000: *University of Minnesota-Duluth* - Processes and Mechanisms of Woodsmoke-induced Immunosuppression. Duluth, MN.

March 2000: **International Symposia on Medaka -** Japanese Medaka: A Sensitive Teleost Model for Assessing the Immunotoxic Effects of Potential Endocrine-Disrupting Chemicals. Osako, Japan.

Nov. 2000: *The Fourth Princess Chulabhorn Science Congress*- Immune System Biomarkers for Predicting the Effects of Environmental Pollution. Bangkok, Thailand.

EDITOR/EDITORIAL BOARD APPOINTMENTS

Editor and Co-Editor:

Metal Toxicology, Co-Editor (Springer Publ.) – (2016)
Pulmonary Immunotoxicology (Klewar Publ.) - (2000)
Immunotoxicology of Occupational and Environmental Metals. (Taylor and Francis) - (1998)
Ecotoxicology: Responses, Biomarkers and Risk Assessment. (SOS Publications) - (1997)
Modulators of Immune Responses: A Phylogenetic Approach - Vol. 2 (SOS Publications)-(1996)
Modulators of Immune Responses - Vol. 1 (SOS Publications) - (1994)
Toxicology and Ecotoxicology News (Taylor & Francis) - (1995-1998)
Book series on: Ecotoxicology (John Wiley & Sons) - (1995-1997)

Associate Editor-

Open Journal of Immunology (2015-2018)
Journal of Developmental Origins of Health & Disease (2012-2013; Themed Editor)
Journal of Toxicology and Applied Pharmacology – (2005-2014)
Journal of Toxicology and Environmental Health - Part A - (2001 - Present)
Biomarkers: Exposure, Effects and Susceptibility - (1995 – 2007)

Editorial Advisory Board-

Envronmental Health Perspectives (2017-2020)
Open Journal of Toxicology (2015-present)
Inhalation Toxicology (2015-present)
Open Journal on Immunology (2009-present)
Journal of Immunotoxicology (2004 - 2016)

Toxicol. Sci. (2007-2016)
Toxicology (1997- 2016)
Environmental Health Perspectives (2009 – 2013; named a top reviewer for 2011)
Environmental Bioindicators (2005- 2011)
Inhalation Toxicology (2004 – 2008; 2013-2016)
Fish and Shellfish Immunology (1997 - 2008)
Toxicology Applied Pharmacology (1996 - 2005)
Diseases of Aquatic Organisms (1995 - 2006)
Aquatic Toxicology (1998 - 2006)
Journal of Toxicology and Environmental Health (1996 - 2001)
Fish Immunology Technical Communications- Vols. 2-5 (1994 - 1997)

CHAIRED SESSIONS/MEETING ORGANIZER (1997 – present, descending order)
<u>*Outside University*</u>

- Organizer/Instructor of International Student & Faculty Workshop on "Fish Immunology" (Tasmania, Australia; February 1997)
- Organizer/Instructor of Student & Faculty Mini-workshop on "Fish Immunology" (Christ Church, New Zealand; February 1997)
- Chairperson at International Meeting on "Developmental and Comparative Immunology" (Williamsburg VA; July 1997)
- Organizer of Student & Faculty International Workshop on "Fish Immunotoxicology Techniques" (American College, Madurai India; February 1999).
- Organizer of Continuing Education Course on "Exposure Assessment: Methods and Applications" at Aquatic Toxicity Workshop Meeting (Edmonton, Canada; October 1999).
- Chairperson of Symposium on "Profiling Immunotoxicology" at Aquatic Toxicity Workshop Meeting (Edmonton, Canada; October 1999).
- International Conference on Environmental and Occupational Lung Disease (Lucknow, India; October, 2000)
- Symposium Coordinator/Chairperson at Society of Toxicology (1993, 1994, 1996-1999; 2005-2009)
- Continuing Education Coordinator/Chairperson at Society of Toxicology (1994, 1995, 2000, 2001)
- Slovenian Society of Toxicology (Nova Gorcia, Slovenia; September 2004, 2005)
- Aerosol Dynamics and Health: Strategies to Reduce Exposure & Harm. (Chairperson, Public Health Issues Involving Environmental & Tobacco Aerosols; Cardiff, Wales 2008)
- SOT - Co-Chair, Symposia and Continuing Education Course, 2009, 2010, 2011, 2015, 2016, 2018, 2019
- ISEE/ISES – co-Chair, Symposia on Environmental Contamination and Indigenous populations. (Ontario, Canada, 2018)

<u>FEDERAL & STATE ADVISORY BOARDS/PANELS/REGULATORY AGENCIES (Contributions to Regulatory Guidelines)</u>

2018-2019: New York City Housing Authority, Advisory Board member for "Healthy Homes".

2017-2018: National Academy of Science, Engineering, Medicine –
-Board on Earth Sciences & Resources; Board on Environmental Studies & Toxicology; Board on Health Sciences Policy: Potential Human Health Effects of Surface Coal Mining Operations in Central Appalachia. 2017-2019.

2015: European Respiratory Society and Environment and Health Committee for American Thoracic Society. Position paper participant on "What constitutes an adverse health of air pollution?" Brussels, BE, March 2015.

2013: American Chemistry Council's Center for Advancing Risk Assessment Science and Policy (ARASP) Workshop - Informing Risk Assessment: Understanding and Communicating Uncertainty in Hazard Assessment. (2013)

2011: Department of Defense
- Gulf War Illness Peer Review Panel (2011)

2013: FDA, Tobacco Control Division, Advisory Consultant (2013)

2013-2006: NASA
- Lunar Dust Exposure Standard Review Panel (2013)
-Lunar Science Institute, Moon Science Grant Review Panel (2008)
- Lunar Dust Non-Advocate Review Panel (Chair, 2006-2008)

2002-2012: National Academy of Science
- National Research Council (NRC): Committee on Low Level Lead in Ammunition (2011 – 2012)
- National Research Council (NRC): Peer Review of NRC Report on Acute Exposure Guideline Levels (2010)
- Institute of Medicine (IOM): Peer Review of IOM Report on Depleted Uranium final document (2008)
- National Research Council (NRC) - Committee on Toxicology/Subcommittee on Spacecraft Water Exposure Guidelines (2001 - 2008)
- Institute of Medicine (IOM): Committee on Gulf War and Health - Part 3 (2002 – 2004)
- Institute of Medicine (IOM): Reviewer for Agent Orange final document (2003)

2012-2010: National Toxicology Program, Science Advisory Board (2010-2012)

1996-2017: National Institute of Health (NIH) & National Institute of Environmental Health Science (NIEHS)

NIEHS, Member reviewer for Core Centers (2018)

-NIEHS, Study Section member (2015-2017)

-NIEHS KO1, K99, R23 reviewer (2014, 2015)
-NIEHS K01, K99 Awards member (2013)
-NIEHS Immunotoxicology Center Program (2012, 2013)
-NIEHS Oceans Centers (2012)
- NIEHS Just-in-time Grants (**Chair,** 2012)
- NIH College of Scientific Reviewers (2010 – 2013)
- NIH Integrative & Comparative Endocrinology (2011)

- NIEHS Time Sensitive Grant (**Chair;** 2010)
-NIEHS P30 (NIEHS Centers of Excellence), (2008, 2009)
- NIEHS Challenge Grants, (2009)
- NIEHS K01 grant applications, (2008)
- NIH Innate Immunity and Inflammation (III) Study Section Full Member, (2005 – 2007)
- NIEHS Program Project grants, *(2006)*
- NIEHS ALTX – 4 (Alcohol and Toxicology) Study Section Full Member, (1996 – 2000)

2005: National Institute of Environmental Health Sciences (NIEHS) & U.S.EPA & NASA
- Expert Panel on "Global Earth Observations: Application to Air Quality and Human Health" (2005)

2005: National Institute of Allergy & Infectious Disease (NIAID) & Department of Defense (DOD)
- Expert Panel Workshop on Pulmonary Threat Agents (2005)

2013-210: New Jersey Department of Environmental Protection
- Human health Committee (2010 – 2013)
- Soil Standards Sub-committee (2010 – 2011)
- Aerosol Sub-committee (2011 – 2012)

2011-2011: United Nations Environmental Program (UNEP) Steering Committee (2006 – 2011)
- Atmospheric Brown Cloud Human Health Panel

2004-2005: U.S. EPA Science Advisory Board & Review Panel
- Metals Risk Assessment Framework Review Panel, (**Co-Chair** of Human Health Breakout Group, 2004 – 2005)
- Nanoparticle Review Panel (2005)

APPOINTMENTS/ELECTED OFFICES
Society of Toxicology (SOT)

Nominating Committee (2018-2020)
Committee for Diversity Initiatives (2014-2015, member; 2015-2016, Co-chair; 2015-2016; Chair, 2016--2017)
Board of Councilors (2011 – 2014**; Secretary-elect**, 2011-2012; **Secretary**, 2012-2014)
Nominating Committee (2007 - 2009)
Congressional Representative (2004 – 2005)
Education Committee (2002 – 2005; **Chair**, 2004 – 2005)
Education Sub-Committee for Minority Initiatives (2001 - 2004; **Chair**, 2003-2004)
Continuing Education Committee (1998 - 2001; **Chair,** 1999 - 2000)
Program Committee (1995-1998)

Inhalation & Respiratory Specialty Section
Councilor (2017-2019)

Ethical and Legal Specialty Section

President (2017-2018)
VP-elect (2016)

Immunotoxicology Specialty Section
President (1999-2000)
Vice-President (1998-1999)
Secretary/Treasurer (1995-1997)
Program Committee (1993-1999)
Awards Committee (1993, 1998, 2000)
Education Committee (Chair, 1992-1996; 2004-2009)
Nominating Committee (1998 - 2001, Chair, 1999-2000)
Councilor (2000-2001)

Metals Specialty Section
President (2003-2004)
Vice President (2002-2003)
Awards Committee (**Chair,** 2001 - 2004)
Program Committee (**Chair**, 2001 - 2004)
Nominating Committee (2001 – 2004, **Chair**, 2001-2003)

MidAtlantic (Chapter) Society of Toxicology (MASOT)
Nominating Committee (2009 [**Chair]**, 2010, 2011)
Past president, Councilor (2009-2010)
President (2008-2009)
Vice President (2007-2008)
Vice President-elect (2006-2007)
Councilor (2001 - 2004)
Program Committee (2000 – Present; Chair 2006-2007)

NYU Langone School of Medicine
Faculty Council Representative (2010-2019; Vice President 2011-2012, 2014-2015);
Benefits and Tenure Sub-committee (2015-2016)
Academic Affairs Sub-committee (Chair, 2012-Present)
Basic Science Sub-committee (co-Chair, 2017-2019)
IACUC Review Board (2009-2011; 2017-2019)
Grievance Committee (2017-2020)
NYU Senate (alternate; 2018-2021)

Department of Environmental Medicine
Promotion & Tenure Committee (2008-2014; **Chair**, 2010-2012)
Search Committee (2010-2013)
Biological Safety Committee- (**Chair**, 1990-1999)
Graduate Steering Committee (1999- 2014; Interim **Co-chair** 2001-2002)
Toxicology Masters' Program (Director, 2002 – 2008; **Co-director**, 2008-2011)

GRANT REVIEWER *Ad hoc* (Federal [Non-NIH]/State/Private):
Federal
Scandanavian Research Program (2013, 2016)
NASA, Moon dust program (2008)
Canadian Centers for Research (2000 – 2004)
DOD (*Ad hoc,* 1999 - present)

EPA (*Ad hoc,* 2002 - present)
Natural Sciences and Engineering Research Council of Canada (*Ad hoc,* 2002 – present)

State/Private
Center for Indoor Air Research
Environmental and Occupational Science Health Inst. (Rutgers U.)
IFS Research Grants for Developing Nations
Johns Hopkins Pilot Projects
Michigan Sea Grant
New Jersey Sea Grant
New York Sea Grant
Philip Morris Foundation

ADJUNCT APPOINTMENTS, CONSULTING, ADVISORY BOARDS

- **Weill Cornell Medical School** (NY, NY) – External Advisory Board for NIH Diversity Grant (2013-2015)
- **Chulabhorn Research Institute & University** (Bangkok, Thailand) **-** Adjunct Professor (2003-present)
- **Cornell University, Inst. for Comparative and Environmental Toxicology** (Ithaca, NY) - Adjunct Professor (1996-2005)
- **American Lung Association** - Criteria Document on Woodsmoke (2001)
- **Fish and Wildlife Services** - Status of the Hudson River (2000)
- **International Life Sciences Institute** - Research strategy on age-related differences in susceptibility (1998)
- **Stratus Consulting Inc. -** Assessment of PCB-contaminated sites (1997 - 2000)

U.S. EPA - Criteria document on the immunotoxicity of endocrine disruptors (1997)

MENTORING ON A GLOBAL LEVEL (6)

- Juliet Igbo (Doctoral student co-mentor – U. of Lagos, Nigeria – 2015-2019)
- Anishka Lewis (Masters student- Jamaica – 2014)
- LeighAnn Koekemoer (Masters student – South Africa-2014)
- Dr. Orish Orisakwe – University of Port Harcourt, Nigeria – 2013-present)
- Dr. Hari Jott Dosih (Nepal Health Research Council – Kathmandu, Nepal- 2014-present)
- Dr. Chanthana Tangjarukij (Chulabhorn Research Institute – Bangkok, Thailand-2012-present)

STUDENT & JUNIOR FACULTY MENTORING
Research Advisor:
***College and High School* (15)**

- Aaron Asiedu-Wiafe (2017-2018; Monroe-Woodbury High School, Monroe, NY)
- Aastha Parikh (2016-2017; Monroe-Woodbury High School, Monroe, NY)
- Daniel Smith (2013-2014; Fairlawn High School, Fairlawn, NJ)
- Alejandro Jorge (2012; Ramapo College, NJ)
- Eric Bloom (2011-2012; Highland Mills High School [Highland Mills, NY])
- Sujay Avencar (2009-2011; Suffern High School [Suffern, NY])
- Sam Openheim (2009-2011; Suffern High School [Suffern, NY])
- Monica Feldman (2007-2009; Spring Valley High School [Spring Valley, NY])

- George Markt (2005-2009; Ramapo High School [Ramapo, NY])
- Payal Roy (2006 – 2007; New York University [NY, NY])
- Rebecca Kurtzman (2005 – 2007; Spring Valley High School [Spring Valley, NY])
- Erica Stone (2006, Ramapo College [Mahwah, NJ])
- Elizabeth Nadziejko (2000; Washingtonville High School [Washingtonville, NY[)
- Kevin Hazard (1999 – 2000; Spring Valley High School [Spring Valley, NY])
- Songeeta Pachachuria (1997-2000; Spring Valley High School, [Spring Valley, NY])
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Post-Baccalaureate (2)
- *Parnavi Desai* (2015-present; NYU, Biology)
- *Tomas Dunne* (2014-2015; Penn State)

***Masters* (30)**
- Arianna Schwartzer (2017-2019; NYU Environ. Health Sci)
- Kathryn Fetce (2016-2018; NYU Environmental Health Sciences)
- Nicholas Lawrence (2016-2018; NYU Environmental Health Sciences)
- Alexander Lucca (2017-2018; NYU Biology)
- Annie J.Thaikkatil (2016-2017; NYU Biology)
- Leena Babiker (2017-2018; NYU Biology)
- Patricia Costa (2014-2016; NYU Environ. Health Sci
- Maria Putilina (2013-2014-NYU, Biology)
- Kirtan Kaur (2013-2015)
- Sarah Attreed (2013-2015)
- Sabina Sutjec (2013-2014-NYU, Biology)
- Kaitlyn Koenig (2012-2014)
- Heather Larkin (2012-2013-NYU, Biology))
- Dana Lauterstein (2011-2013) – 2 SOT student awards (2013)
- Yi-Chuh Chen (2010-2011 Incomplete-NYU Biology)
- Ya-Chien Yu (2010-2011-IncompleteNYU Biology)
- Yuan-Chun Hsiao (2010-2011-Incomplete NYU Biology)
- Lauren Rosenblum (2009-2011-NYU Biology)
- Sandra Perella (2008-2010)
- Kotaro Hoshido (2007-2009-NYU Biology)
- Jacqueline Grabowski (2006-2008)
- Elizabeth Vanza (2004 – 2006) – *SOT student award (2006)*
- Elizabeth Berg (2003 - 2005)
- Shannon Doherty (2002 - 2005)
- Colette Prophete (1998 - 2001)
- Jessica Duffy (1999 - 2001)
- Migali Jorge (1998 - 2000)
- Cheryl Premdass (1998 - 2000)
- Andrea Raymond (1997 - 2000) – *1 SOT award*
- Thomas McManus (1994 – 1996, Co-advisor)

Doctorate **(9)**

- Pamela Tijerna (2013-present) – *SOT CDI award (2014); SOT (1st place Hispanic Organization of Toxicology, 2015); SOT(Mary Amdur Inhalation Fellowship, 2015)*
- Dana Lauterstein (2013-present)- SOT (Safety Assessment Specialty Section, 2015)
- Juliett Igbo (2015-2016), Co-Advisor (U. of Lagos, Nigeria)
- Sheung Pui Ng (2004 - 2010) – *9 SOT student awards including Novartis Achievement Award (2008-2010)*
- Jessica Duffy (2001 – 2007) – *2 SOT awards (2004); 3 SETAC awards (2004, 2005, 2006)*
- Chanthana Settachan (Co-Advisor; 2003 – 2009; Chulabhorn Research Institute, Bangkok Thailand)
- Erik Carlson (1999- 2003) – *1 SOT award (2000)*
- Ninah Enane (Co-Advisor, 1995 - 1999)
- Peter Atkins (Co-Advisor, 1992 - 1996)

Post-doctoral Trainees **(2) & Mentoring Committees**

- Jason Blum (2009 – 2012) – *1 SOT post-doc award*
- Daniel Willis (2011 – 2013)- NSF/FDA post-doctoral fellowship (Zelikoff, PI)-2013

Junior Faculty Mentoring Committee **(2)**

- Jason Blum (2012 – Present)
- Kevin Cromar (2012-Present)

<u>**Doctoral Thesis Committee** (12):</u>

- Kirtan Kaur (2016-2018, Chair)
- Carolyn Klocke (2015-2017) – University of Rochester (External Examiner)
- Mary Francis (2015-2016) - Rutgers University (External Examiner)
- Eric Saunders (2012-2015)
- Joshua Vaughn (2012 – 2015)
- AJ Cuevas (2007 – 2012)
- Jessica Lyon (2007 - 2012)
- Judy Blatt Nichols (Chair, 2007 – 2011)
- Patricia Gillespie (2006 - 2010)
- Elizabeth Vanza (Chair, 2004 – 2009)
- Ann Zulkosky (2005 – 2007; SUNY Stony Brook)
- Samantha DeLeon (Chair, 1999 – 2003)

COMMUNITY OUTREACH, EDUCATION & ENGAGEMENT INITIATIVES:

- ***Director****, Community Outreach & Education Program, NYU, Dept. of Environ. Med.* (2005- present)
- ***Director****, NIEHS Center of Excellence, Community Outreach & Engagement Program, NYU, Dept. of Env. Med. (2005 – present)*
- ***Director,*** *NIEHS Superfund Community Outreach and Education Core, NYU, Dept. of Environ. Med.* (2005- 2010)

- ***Co-director***, NIEHS Superfund Translation Core, *NYU, Dept. of Environ. Med.* (2005- 2011)

  Community Partners:
  - *Ironbound Community Corporation (ICC): Newark, NJ (2015-present)*
  - *Ramapough Lenape Tribal Nation: Ringwood, NJ/Mahwah, NJ/Hillburn, NY (2013-present)*
  - *City of Garfield, NJ (2012-present)*
  - *Susquehanna, PA: Fracking communities (2015-2016)*
  - Flint, Michigan via Water Defense

Translation/Communication of toxicology to non-toxicologists & underserved minorities

- Community groups in PA and NY: Environmental and Health Implications of Hydraulic Fracturing (2013-2014).
- Ramapo Indians: Living on a Superfund Site (2014-present)
- NY Presbyterian Lang Program for Underserved Youth (2010 - Present)
- *Harlem Children Society Mentoring Program - Bronx, NY* (2010-Present)
- Y-2 Kids (NY State 4th – 12th grade, Career day representative, 2008 - Present)
- *Center for Talented Youth, New York University Department of Environmental Medicine & Johns Hopkins Center for Talented Youth (2005 – Present)*
- *Environmental Commission of Ramsey (2001 – 2007; Vice-Chair; 2004-2006)*

- Ramsey, New Jersey. Woodburning: A Cozy Atmosphere or a Public Menace? (2003)

- *Senior Citizen Advisory Board of Ramsey* (2003 - 2005)
- *Ramsey High School (Presenter on toxicology and the environment 2005-2006)*
- *Youth Guidance Commission of Ramsey* (1999 - 2001)
- *Rotary Club*, Goshen, New York. Woodburning: A Cozy Atmosphere or a Public Menace? (2003)
- *Upper Saddle River Community Center*, Upper Saddle River, New Jersey. The Hazards of Woodburning (1997)

Non-Academic Related Outreach Committees:

- 2011- 2014 – *Board of Ethics*, Community Hospice of Bergen County (NJ)
- 2009- 2014 – *Fundraising Committee*, Community Hospice of Bergen County (NJ)
- 2006-2013 – President, Condominium Association
- 2013-2016 – Vice-President, Condominium Association
- 2018 – South Bronx Asthma Coalition

Exhibit B

MATERIALS AND DATA CONSIDERED

Literature

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Depositions

Deposition of Alice M. Blount Dated 4.13.2018
Deposition and Exhibits of Laura M. Plunkett Dated 1.11.2017-1.13.2017
Deposition of Dr. Thomas Dydek Dated 8.21.18
Deposition and Exhibits of John Hopkins Dated 8.16.18-8.17.18
Deposition and Exhibits of Julie Pier Dated 9.12.18-9.13-18
Deposition and Exhibits of Pat Downey Dated 8.7.18-8.8.18
Deposition of Robert Glenn Dated 10.18.18

Deposition and Exhibits of Donald Hicks Dated 6.28.18-6.29.8

Reports

Expert Report of Michael M. Crowley, PhD
Expert Report of William E. Longo, PhD and Mark W. Rigler PhD. Analysis of J&J Baby Powder & Valiant Shower to Shower Talc Products for Amphibole (Tremolite) Asbestos Expert Report. August 2, 2017.
Expert Report of William E. Long, PhD, Mark W. Rigler, PhD and William B. Egeland, M.S., P.G. Below the Waist Application of J&J Baby Powder Expert Report. September, 2017.
Expert Report of William E. Longo, PhD and Mark W. Rigler PhD. TEM Analysis of Historical 1978 Johnson's Baby Powder Sample for Amphibole Asbestos . February 16, 2018.
Expert Report of William E. Longo, PhD and Mark W. Rigler, PhD. November. 14, 2018.
Expert Report (Brower v. J&J) of Dr. Thomas Dydek
Expert Report (Brower v. J&J) of Dr. Laura Plunkett
Supplmental Expert Report (Brower v. J&J) of Dr. Laura Plunkett